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# HENRY FORD HOSPITAL MEDICAL BULLETIN

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# PLASMA FIBRINOGEN AND SURGICAL TRAUMA

C. Paul Hodgkinson, M.D.,*
Paul W. Pifer, M.D.* and
Donald G. Remp, Ph.D.**

This is a preliminary report on a project to determine the influence of surgical trauma upon plasma fibrinogen. Observations upon fifteen patients are recorded.

Using serial blood samples, a biphasic fibrinogen response was observed. This was characterized by a brief period of fibrinogen depression followed by a rather prolonged phase of hyperfibrinogenemia. There was some indication that the quantitative fibrinogen changes could be correlated with the severity of surgical trauma.

The surgical procedures used for the fifteen gynecological patients were graded for the degree of surgical trauma. Such relative factors as the length of the incision, violation of tissue spaces, crushing and clamping of tissues, duration of the operation, and the surgeons estimation of the amount of effort expended constituted the qualifying points considered important.

With these grading points in mind it was possible to divide the fifteen patients into four groups of increasing degree of surgical trauma. The following outline gives the details used for evaluation.

| Group | Number of Patients | Surgical procedures and descriptive designation of the group. "A" indicates the group of least trauma and "D" the group of maximum trauma. |
|---|---|---|
| A | 5 | "Dilatation and Curettage operations"—included patients numbered from 1 through 5. |
| B | 4 | "Incision operations"—The essential feature of this group was the making and closing of a surgical incision without extirpation of a major amount of tissue or of an organ. Includes patients numbered 6 through 9. |
| C | 4 | "Extirpation operations"—The requirement for this group was complete hysterectomy. Included were patients numbered 10 through 13. |
| D | 2 | "Radical extirpation operations"—Included two patients subjected to radical surgery for malignancy, numbered 14 through 15. |

## *METHOD OF STUDY*

The plan of study was designed to insure uniform procedure. From experience

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with obstetrical patients; it was deemed wise to consider plasma fibrinogen as an extremely labile substance. Because we were probably seeking fibrinogen change, of subclinical importance, the plan of study was devised to detect abrupt, if minors fluctuations in quantitative plasma fibrinogen.

On the day of surgery, blood samples were obtained each one-half hour during the operation and then at two hour intervals for the next seven hours. Except for the day of operation, daily pre- and postoperative blood samples were obtained.

To equalize the time factor, all blood samples collected in any one day were stored in a refrigerator until the following morning.

In the laboratory all blood samples received in any one day were analyzed by one technician.* The Wu-Ling method, with an estimated normal range of from 200 to 400 mg. % was employed.

All blood samples were obtained by one physician and consisted of ten cc. of venous blood mixed with two drops of a saturated solution of potassium oxalate. An incident emphasized the importance of clean, uncomplicated venipuncture in procuring blood for plasma fibrinogen. Patient No. 15 (Group D) was obese and difficulty was encountered in obtaining one sample. Prior to entering the vein wall tissue juice was observed in the barrel of the syringe. Fearing thromboplastin contamination, a duplicate blood sample using a new apparatus was obtained with an initially successful venipuncture. The first speciman was reported as containing 143 mg. % fibrinogen while the second specimen contained 207 mg. %. The last value compared favorably with other values for this patient.

*RESULTS:*

The "Dilatation and Curettage operation" group consisted of five patients. One patient was operated upon because of incomplete abortion (patient No. 1). The other four were suffering from menorrhagia, and the procedure was diagnostic. Replacement of blood was necessary for patients No. 4 and 5 because of severe blood loss anemia. Patient No. 4 received 1,000 cc. of bank blood on the day before operation and 500 cc. of bank blood during the operative procedure.

Patient No. 5 received 500 cc. of bank blood the day before operation and 500 cc. during operation. Inhalation anesthesia was employed for patients in Group A. The fibrinogen results are listed in Chart No. 1, and the average values for the group are plotted in Figure No. 1.

Except for patients numbered 4 and 5, significant plasma fibrinogen changes were not observed. Both of these patients were the recipients of considerable quanitites of bank blood. Upon admission to the hospital the hemoglobin value for patient No. 4 was 5.6 grams percent and for patient No. 5, 8.9 grams percent. One might expect some change in quantitative plasma fibrinogen as the result of transfusion, but it is difficult to reconcile plasma fibrinogen depression on a basis of blood replacement alone.

*This phase of the work was supervised by Dr. Victor Schelling, Division of Physiological Chemistry, Department of Laboratories.

## GROUP B

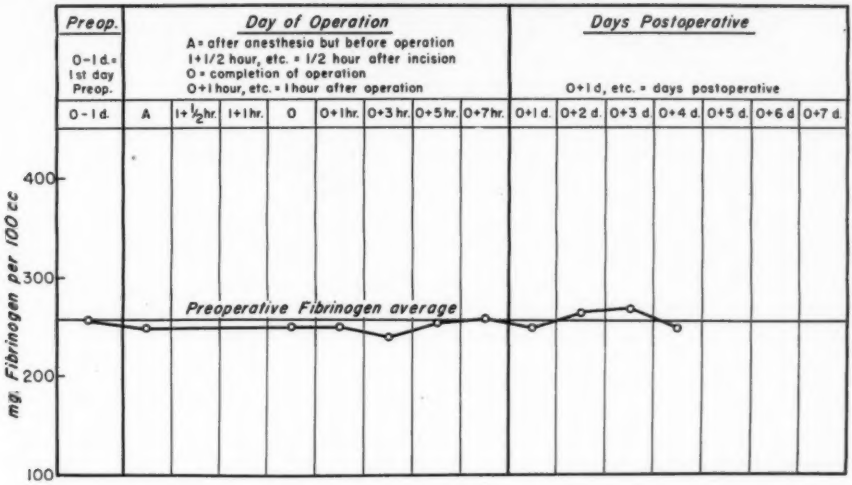
The "incision operation" group was composed of four patients subjected to a variety of surgical procedures. Except for patient No. 6 whose operation consisted of biopsy of a small area of chronic cystic mastitis, no major amount of tissue was excised from patients of this group. The operative procedure characterizing Group B consisted of the making and closing of a surgical incision without extirpation of a major amount of tissue or of an organ. Some controversy in this respect might develop over patient No. 7 who was operated upon under local infiltration anesthesia with 2% xylocaine for elective cesarean section. For this report, we have not considered the delivered infant to constitute "excised tissue." Patient No. 8 was operated upon for exploration of the right kidney, and patient No. 9 for repair of an enterocele and revision of plastic repair of the left ureter. No patient of this group was the recipient of a blood transfusion.

CHART NO. 1

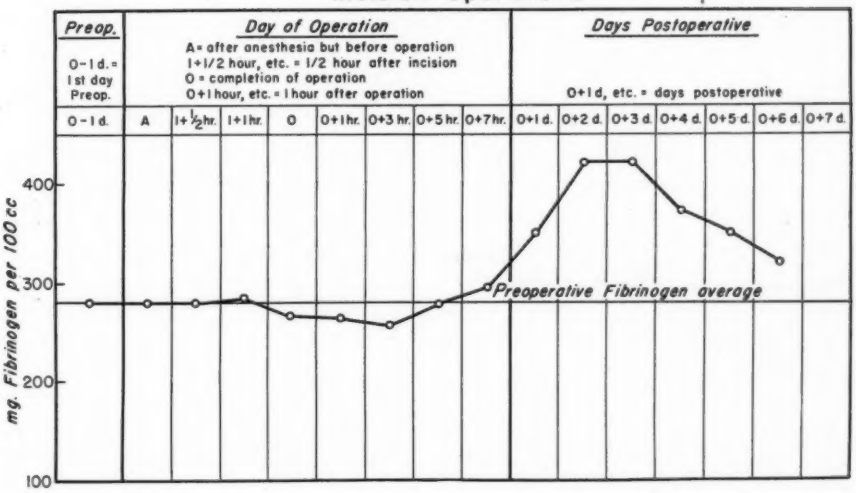
| TIME OF COLLECTION OF BLOOD SAMPLE AND DESIGNATION OF SAMPLE | Pt. #1 | Pt. #2 | Pt. #3 | Pt. #4* | Pt. #5* | Average |
|---|---|---|---|---|---|---|
| 1. Day before operation (0 minus 1 day) | 289mg.% | 249mg.% | 251mg.% | 273mg.% | 220mg.% | 256mg.% |
| 2. After induction of anesthesia (A) | 276 " | 231 " | 255 " | 297 " | 173 " | 246 " |
| 3. ½ hour after start of operation (I plus ½ hour) | | | | | | |
| 4. 1 hour after start of operation (I plus 1 hour) | | | | | | |
| 5. Termination of operation (0) | 285 " | 231 " | 248 " | 279 " | 195 " | 248 " |
| 6. 1 hour after termination of operation (0 plus 1 hour) | 290 " | 258 " | 246 " | 267 " | 175 " | 247 " |
| 7. 3 hours after termination of operation (0 plus 3 hours) | 276 " | 245 " | 247 " | 255 " | 182 " | 241 " |
| 8. 5 hours after operation (0 plus 5 hours) | 296 " | 238 " | 243 " | 267 " | 220 " | 253 " |
| 9. 7 hours after operation (0 plus 7 hours) | 285 " | 244 " | 253 " | 255 " | clot | 259 " |
| 10. 1 day after operation (0 plus 1) | 310 " | 241 " | 254 " | 252 " | 190 " | 249 " |
| 11. 2 days after operation (0 plus 2) | 297 " | 240 " | 278 " | 269 " | 246 " | 266 " |
| 12. 3 days after operation (0 plus 3) | 309 " | 240 " | | 262 " | | 270 " |
| 13. 4 days after operation (0 plus 4) | | 249 " | | | | |

*Designates patients receiving blood transfusions.

## Group – A
## Dilatation and Curettage


Preop.
0 – 1 d. = 1st day Preop.
Day of Operation
A = after anesthesia but before operation
1 + 1/2 hour, etc. = 1/2 hour after incision
O = completion of operation
O + 1 hour, etc. = 1 hour after operation
Days Postoperative
O + 1 d, etc. = days postoperative
0 – 1 d.
A
1 + ½ hr.
1 + 1 hr.
O
O + 1 hr.
O + 3 hr.
O + 5 hr.
O + 7 hr.
O + 1 d.
O + 2 d.
O + 3 d.
O + 4 d.
O + 5 d.
O + 6 d.
O + 7 d.
mg. Fibrinogen per 100 cc
400
300
200
100
Preoperative Fibrinogen average

## Group – B
## Incision Operations


Preop.
0 – 1 d. = 1st day Preop.
Day of Operation
A = after anesthesia but before operation
1 + 1/2 hour, etc. = 1/2 hour after incision
O = completion of operation
O + 1 hour, etc. = 1 hour after operation
Days Postoperative
O + 1 d, etc. = days postoperative
0 – 1 d.
A
1 + ½ hr.
1 + 1 hr.
O
O + 1 hr.
O + 3 hr.
O + 5 hr.
O + 7 hr.
O + 1 d.
O + 2 d.
O + 3 d.
O + 4 d.
O + 5 d.
O + 6 d.
O + 7 d.
mg. Fibrinogen per 100 cc
400
300
200
100
Preoperative Fibrinogen average

Although blood volume studies have not been performed, blood loss was not considered excessive in any patient of this group. As part of the anesthetic routine, each patient received a slow infusion of 5% glucose in water during the surgical procedure to facilitate the administration of indicated intravenous therapy during surgery. The possible influence of this procedure on fibrinogen behavior is a project for future investigation.

The fibrinogen behavior of patients of Group B is recorded in Chart No 2 and the average is graphically shown in Figure No. 2.

In this group of patients the hypo-hyper, biphasic fibrinogen response was clearly evident. Because the operative time for patient No. 6 was too short to obtain blood samples No. 3 and No. 4, these values were calculated from averaging the two previous fibrinogen values. This liberty was taken to avoid distortion caused by the high normal fibrinogen concentration observed in patient No. 9.

Upon observation of the group averages, it was evident that the hypofibrinogen phase was of short duration. Its onset began during the surgical procedure and was maximum 3 hours after termination of the operation. The maximum fibrinogen depression averaged 8%. A swing to hyperfibrinogenemia was evident beginning with the five hour postoperative blood specimen and was maximum on the third postoperative day when a peak of 421 mg. % was reached. Observations were discontinued on the sixth postoperative day and at that time the fibrinogen values had not reached the preoperative level. The average increase in plasma fibrinogen was slightly more than 50% over the preoperative level.

In considering some of the reasons for the observed fibrinogen behavior, the effect of general anesthesia as a factor of importance can be eliminated from the results observed in patient No. 7. She received no general anesthetic and only a minimal amount of preoperative sedation (1/150 grain of scopolamine was administered ½ hour preoperatively). The fibrinogen response was as extensive as that observed in any of the other patients of this group.

## GROUP C

Four patients subjected to complete hysterectomy were placed in category C—"Extirpation operations." The vaginal approach was used for patient No. 13 and the abdominal approach for the others.

Patient No. 12* presented a unique problem. She was approximately 28 weeks pregnant and demonstrated signs of rupture of a uterine cesarean scar. Upon opening the abdomen, viable twin babies were found free in the peritoneal cavity. The placenta remained partially attached to the uterine surface. On the anterior surface of the uterus was a large defect bounded by a ring of fibrous tissue which showed only slight evidence of trauma. Only slight bleeding occurred from the placental site which was covered by a hematoma. Apparently the membranes had ruptured intraperitoneally resulting in the infants being delivered through this site of chronic uterine rupture. Of interest for this study was the volume of blood administered to this patient. Because of the ominous nature of the pre-

*Patient of Dr. Walter C. Rattan.

## CHART NO. 2

| TIME OF COLLECTION OF BLOOD SAMPLE AND DESIGNATION OF SAMPLE | Pt. #6 | Pt. #7 | Pt. #8 | Pt. #9 | Average |
|---|---|---|---|---|---|
| 1. 0 minus 1 day | 261mg.% | 275mg.% | 262mg.% | 327mg.% | 281mg.% |
| 2. A | 242 " | 280 " | 238 " | 365 " | 281 " |
| 3. I Plus ½ hour | (252)* " | 284 " | 225 " | 365 " | 291 " (281)** |
| 4. I plus 1 hour | (252)* " | 288 " | 250 " | 350 " | 296 " (285)** |
| 5. O | 266 " | 257 " | 235 " | 314 " | 268 " |
| 6. 0 plus 1 hour | 246 " | 257 " | 253 " | 305 " | 265 " |
| 7. 0 plus 3 hours | 216 " | 270 " | 250 " | 298 " | 258 " |
| 8. 0 plus 5 hours | 250 " | 274 " | 277 " | 333 " | 280 " |
| 9. 0 plus 7 hours | 189 " | 261 " | 292 " | 349 " | 273 " |
| 10. 0 plus 1 days | clot | 303 " | clot | 399 " | 351 " |
| 11. 0 plus 2 days | 275 " | 433 " | | 419 " | 376 " |
| 12. 0 plus 3 days | | 433 " | | 410 " | 421 " |
| 13. 0 plus 4 days | | 347 " | | 402 " | 374 " |
| 14. 0 plus 5 days | | 327 " | | 373 " | 350 " |
| 15. 0 plus 6 days | | 320 " | | 318 " | 319 " |

**Average includes calculated values for patient No. 6.

*Values were calculated from average of two preceding values.

## CHART NO. 3

| TIME OF COLLECTION OF BLOOD SAMPLE AND DESIGNATION OF SAMPLE | Pt. #10 | Pt. #11* | Pt. #12* | Pt. #13 | Average |
|---|---|---|---|---|---|
| 1. 0 minus 1 day | 226mg.% | 312mg.% | 280mg.% | 262mg.% | 270mg.% |
| 2. A | 245 " | 254 " | 285 " | 257 " | 260 " |
| 3. A plus ½ hour | 202 " | 260 " | 273 " | 257 " | 248 " |
| 4. A plus 1 hour | clot | clot | 246 " | 253 " | 249 " |
| 5. O | 215 " | 262 " | 240 " | 250 " | 242 " |
| 6. 0 plus 1 hour | 208 " | 233 " | 252 " | 249 " | 235 " |
| 7. 0 plus 3 hours | 220 " | 243 " | 293 " | 244 " | 250 " |
| 8. 0 plus 5 hours | 220 " | 261 " | 305 " | 235 " | 255 " |
| 9. 0 plus 7 hours | 224 " | 248 " | 312 " | 226 " | 253 " |
| 10. 0 plus 1 day | 276 " | 280 " | 308 " | 312 " | 294 " |
| 11. 0 plus 2 days | 332 " | | 312 " | 276 " | 307 " |
| 12. 0 plus 3 days | | | 332 " | 418 " | 375 " |
| 13. 0 plus 4 days | | | 332 " | | 332 " |
| 14. 0 plus 5 days | 332 " | | 312 " | | 322 " |
| 15. 0 plus 6 days | 309 " | | 335 " | | 322 " |
| 16. 0 plus 7 days | 262 " | | 323 " | | 293 " |
| 17. 0 plus 8 days | 266 " | | 310 " | | 288 " |
| 18. 0 plus 9 days | 280 " | | | | 280 " |

*Blood transfusion administered during surgery.

operative diagnosis, the volume of blood given this patient was 1500 cc. This amount greatly exceeded the estimated blood loss and the patient never demonstrated clinical evidence of shock. Nevertheless, her fibrinogen reaction followed the pattern of the other patients of Group C. Such evidence is rather convincing that blood loss incident to surgery is not a major factor in causing the observed fibrinogen alterations.

The results are shown in Chart No. 3 and Figure No. 3.

In Group C, maximum fibrinogen depression was evident in blood specimen, 0 plus 1 hours, having fallen from 270 mg. % to 235 mg. %, or 13%. There appeared to be a delay for plasma fibrinogen to rise as observed through blood specimen, 0 plus 7 hours, which averaged only 253 mg. %.

Hyperfibrinogen values were clearly evident from the first postoperative day. The average remained slightly elevated through the eighth postoperative day. The maximum elevation to 375 mg. % occurred on the third postoperative day, which was 39% above the preoperative plasma fibrinogen value.

## GROUP D

"Radical extirpation operations." Group D was composed of two patients subjected to radical surgery for malignancy. Patient No. 14 was subjected to radical mastectomy and Patient No. 15 to radical hysterectomy. Patient No. 14 was incompletely studied because malignancy of the breast was not suspected until after microscopic examination of the frozen section. As a result, averages have no statistical meaning for this group but are shown for the sake of uniformity.

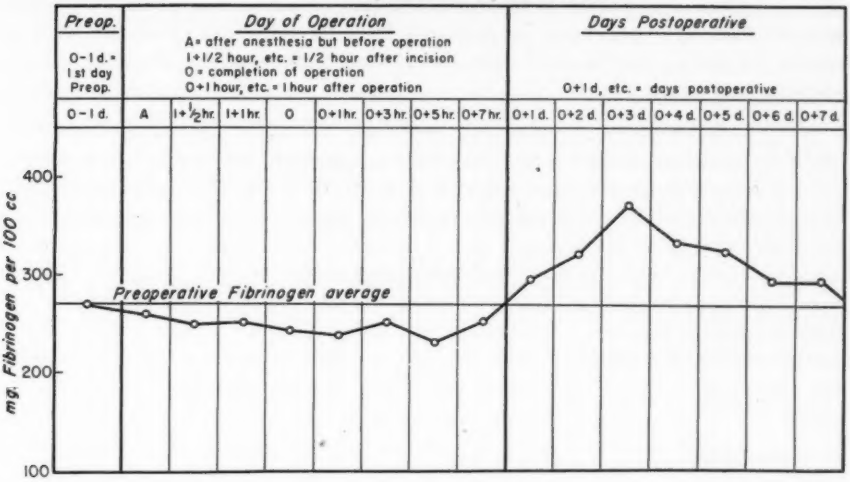
In patient No. 15, the fibrinogen depression phase is maximum upon termination of the operation, having fallen from 240 mg. % to 207 mg. %—a fall of slightly move than 13%. Again there appeared to be a delay in the fibrinogen rise. Hyperfibrinogenemia was not apparent until the first postoperative day.

The phase of fibrinogen elevation in Group D demonstrated a pattern quite different from that observed in the other groups. When studies were discontinued on the seventh postoperative day, the fibrinogen level was still rising. This observation was consistent with the work of others who have shown that fibrinogen elevation occurs as the result of inflammation.[1] In patients subjected to radical procedures, healing is prolonged and frequently complicated. The fibrinogen elevation averaged more than 56%. Blood replacement therapy was administered to both of these patients. (Chart No. 4 and Figure No. 4)

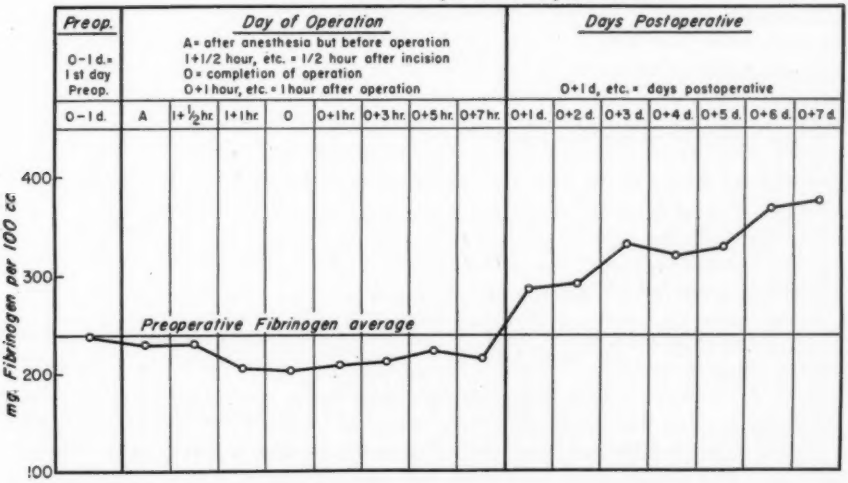
*COMMENT:*

Fibrinogen values vary considerably both in health and disease. The normal range, using the Wu-Ling[2] method varies from 200 to 400 mg. %.[3] In conjunction with the present study, the nonpregnant preoperative plasma fibrinogen value averaged 267 mg. %. A similar study[4] conducted in pregnancy showed the predelivery average to be 315 mg. %. Plasma fibrinogen can be elevated by ingestion of a high protein diet. Complete absence of plasma fibrinogen has been reported as a congenital condition.

## Group – C
## Extirpation Operations




## Group – D
## Radical Extirpation Operations



According to Smith et al[5] fibrinogen values are elevated in acute bacterial respiratory infections, postoperative states, dicumarolized states, acute pancreatitis, untreated malignancies, and radiologically treated malignancies. These authors state that fibrinogen is depressed by viral infections. Others[6] have reported increased plasma fibrinogen in various inflammatory diseases, arteriosclerosis and eclampsia.

Some pathological conditions cause depressed fibrinogen values. In obstetrics, abruptio placentae, retention of a dead fetus and possibly automatic fluid embolism cause critically low fibrinogen values.[7, 8, 9, 10, 11, 12] Typhoid fever, cachexia, chronic disease, liver dysfunction, nephritis, bone marrow disease and acute

CHART NO. 4

| TIME OF COLLECTION OF BLOOD SAMPLE AND DESIGNATION OF SAMPLE | Pt.#14* | Pt.#15* | Average |
|---|---|---|---|
| 1. 0 minus 1 day | | 240mg.% | 240mg.% |
| 2. A | | 233 " | 233 " |
| 3. A plus ½ hour | | 235 " | 235 " |
| 4. A plus 1 hour | | 207 " | 207 " |
| 5. O | 202mg.% | 207 b (142 a) | 205 " |
| 6. 0 plus 1 hour | 211 " | 210 " | 211 " |
| 7. 0 plus 3 hours | clot | 214 " | 214 " |
| 8. 0 plus 5 hours | 234 " | 214 " | 224 " |
| 9. 0 plus 7 hours | 209 " | 226 " | 218 " |
| 10. 0 plus 1 day | 290 " | 278 " | 284 " |
| 11. 0 plus 2 days | 336 " | 252 " | 294 " |
| 12. 0 plus 3 days | 336 " | 325 " | 331 " |
| 13. 0 plus 4 days | 296 " | 346 " | 321 " |
| 14. 0 plus 5 days | 296 " | 352 " | 324 " |
| 15. 0 plus 6 days | | 369 " | 369 " |
| 16. 0 plus 7 days | | 375 " | 375 " |

*Blood transfusion administered during surgery.

hemorrhage have been reported as fibrinogen depressing conditions.[13] Coon and Hodgson,[14] and Mathes[15] reported fibrinogen depression, abnormal fibrinolysis, and uncontrollable hemorrhage following pulmonary surgery and shock. Bennicke and Mullertz[16] suggested that most instances of acquired fibrinogenopenia were associated with various destructive processes.

Fibrinogen depression has been the object of considerable experimental effort during the past ten years.[17, 18] Thromboplastin studies by Schneider,[7, 8] Page and others [19, 20, 21, 22, 23] have added considerably to the understanding of fibrinogen physiology. The fibrinogen deficiency observed in obstetrics has been generally accepted as being the result of thromboplastin gaining entrance into the blood stream. Fibrinogen depression has also been reported following injection of trypsin[24] and ACTH and/or cortisone.[25, 26] Innerfield working with trypsin

produced afibrinogenemia in dogs. It is not known if this effect was caused by the lytic properties of trypsin, or if this was caused by a thromboplastin influence resulting from contamination of the blood stream. ACTH was studied by Fearnley et al.[26] They tested seven normal subjects after injection of 40 mg. ACTH. Four of the group showed depression, two showed elevation and one showed no change in plasma fibrinogen. Fearnley et al could not conclude if this reaction was the result of the physiological effect of ACTH or if it was a foreign protein reaction.

The values of plasma fibrinogen have been related to trauma and inflammation. Foster and Whipple[1] in 1922 demonstrated hyperfibrinogenemia following trauma and attributed the response to the process of inflammation. Postoperative hyperfibrinogenemia was reported by Chanutin et al,[17] and Henriques et al.[18] Hidalgo, Rowell and Ralls[27] reported upon the plasma fibrinogen response to tissue trauma in rabbits. Using a serial schedule of blood sampling similar to the one employed in the present report, Hidalgo demonstrated biphasic, hypo-hyper fibrinogen reactions following maximal trauma. They considered the thromboplastin theory to explain most satisfactorily the phenomena observed. Hidalgo et al also suggested that minute amounts of thromboplastin possibly caused the liver to manufacture more fibrinogen to account for the phase of hyperfibrinogenemia.

Coon and Hodgson[14] and Mathes[15] ascribed fibrinogen depression to abnormal fibrinolysis. In a recent study correlating plasma fibrinogen and fibrinolysis to labor and delivery, we were unable to demonstrate that increased fibrinolysis had any influence upon plasma fibrinogen values. However, if fibrinogen is depressed to the critical level of 90 mg. %,[12] fibrinolytic activity can be easily demonstrated on the soft clot formed under such circumstances. It has been observed that elevation of the fibrinogen level to more normal values promptly prevents clinical fibrinolytic activity. From these observations, it appears that the fibrinogen depressing ability of natural occurring fibrinolysins has yet to be proved.

According to the thromboplastin postulate of Schneider and others, defibrination of plasma occurs when thromboplastin gains entrance into the blood stream. Prothrombin is converted to thrombin which influences fibrinogen to be converted to fibrin. Fibrin is precipitated to an insoluble net-like jel which coats the endothelial surfaces and occludes the smaller capillaries as fibrillar deposits. Page et al[29] have demonstrated that tissue thromboplastin injected into any vein reaches all organs and is not filtered out by the lungs.

Also, in accordance with this theory, the massiveness of the reaction is reflected by the degree of immediate plasma fibrinogen depression.

Serial fibrinogen studies previously reported on cases of obstetrical hypofibrinogenemia demonstrated that plasma fibrinogen depression was an extremely transient phenomena. One patient demonstrated fibrinogen regeneration from zero percent to 78.4 mg. % in 105 minutes while the plasma fibrinogen in one other patient rose from zero percent to 87.5 mg. % in 130 minutes.[12]

Accordingly, the potentialities for this morbid process to produce clinical symptoms is twofold. Shock may occur if the capillary bed is occluded. Hemorrhage will result if depression below the critical level of 90 mg. % of plasma fibrinogen is

exceeded. The realization that the circulatory system may be flooded with insoluble fibrin following surgical procedure and trauma is a sobering thought. Its relationship to the etiology of thromboembolic phenomena, shock, sludged blood, obscure hemorrhage and such specific clinical conditions as acute hemorrhagic pancreatitis, burns, and crush injuries will require further investigation.

## CONCLUSIONS

1. Serial plasma fibrinogen studies performed upon 15 gynecological patients demonstrated that a biphasic fibrinogen reaction occurred as the result of surgical trauma.

2. The first reaction was plasma fibrinogen depression. This was followed by a period of hyperfibrinogenemia.

3. There was a suggestion that the intensity of the biphasic reaction varied directly with the degree of surgical trauma.

4. The possible mechanisms for its production are considered.

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# ARTHRITIS OF THE TEMPOROMANDIBULAR JOINT DIAGNOSIS AND MANAGEMENT*

DON H. BELLINGER, D.D.S.**

The fundamental principles of temporomandibular joint disease have been rather slow in coming into focus. There have been a number of reasons, acting singly or in combination, for this delay.

The early literature dealing with this subject was based on clinical impressions that were not always valid. This misinformation was repeated and quoted in subsequent articles until accepted as factual but eventually proved to be in error.

There has also been delay in applying to the temporomandibular joint knowledge in general that applies to diseases of other joints. This delay was undoubtedly due to the lack of knowledge on the part of the dental profession of arthritis in general and lack of interest on the part of the physician for this particular part of the anatomy.

Disease of joints is termed arthritis in whatever form it may occur. It is a part of that large symptom complex known as Rheumatic Disease in which pain and stiffness of some portion of the musculo-skeleton system is prominent. Arthritis is the term used when the joints themselves are the major seat of the pathology. It is a disease of connective tissue and is one of the oldest known yet most neglected diseases.

Technically, all locations in the skeleton where bone ends are joined together are joints. Thus, the suture lines of the skull are joints. The articulations between the bodies of the vertebrae represent another type of joint in which the bone surfaces are separated by a cushion of fibrocartilage and are joined and maintained by dense, fibrous bands or ligaments, allowing for some, but little, movement. Diarthrosis is the name applied to joints that permit free movement. The diarthrodial joint differs from the above-mentioned types in that the ends of the bone are covered with articular cartilage, which is bathed by a mucinous fluid, the synovial fluid, so named because of its superficial resemblance to egg-white. The joint cavity in dysarthrodial joints is enclosed by a sleeve of loose connective tissue—the synovial membrane. This layer of tissue is surrounded and supported by a capsule of fibrous connective tissue, reinforced by ligaments. In certain of the joints, such as the knees and the temporomandibular joints, there is a partial or complete layer of fibrocartilage between the articular cartilages.

### *Normal Anatomy of the Temporomandibular Joints*

The temporomandibular joint, or the articulation between the mandible and the cranium, is a complex joint because an articular disc is interposed between

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the temporal bone and the condylar head of the mandible, dividing the articular space into an upper and lower compartment. The condyle is strongly convex in an anterior-posterior direction and slightly convex medio-laterally. The articulating surface of the condyle faces upward and anteriorly so that in a lateral view the neck of the mandible seems to be bent forward. The articulating surface of the temporal bone is composed of the concave articular fossa and the convex eminentia articularis, or tubercle. The degree of convexity of the eminentia articularis is highly variable.

The meniscus, or intra-articular disc, is an oval structure of dense, fibrous connective tissue interspersed with a few cartilaginous cells. In the middle it is thin, while anteriorly and posteriorly it is considerably thicker, the posterior portion being especially thick. Posteriorly the disc continues into a thicker layer of loose and vascularized connective tissue. The joint is enclosed by a sleeve of loose tissue and the disc is attached to this sleeve of tissue in its entire circumference. This sleeve of loose connective tissue, known as the joint capsule, is attached above to the rim of the glenoid fossa and below to the neck of the condyle, to which it is attached farther down posteriorly than anteriorly. The inner surface of this joint capsule is the synovial membrane which forms small folds and villa and is richly supplied with blood vessels, nerves and lymphatics, a prerequisite for the function of a synovial membrane, namely, to furnish the lubrication and nutritional requirements to the joint cartilage. It has never been demonstrated that the lymphatics of the synovium communicate with the joint spaces directly, but no apparent barrier separates them. Thus it appears that the joint space is not a true body cavity, but more accurately, a tissue space. This point is of importance in the consideration of injection therapy. Surrounding and supporting the synovial membrane is a fibrous connective tissue that completes the joint capsule. This capsule is very lax between the temporal bone and the disc, which allows for the gliding movement of the joint, but it is much tighter between the disc and the mandibular condyle in which the hinging action of the joint largely takes place.

The articular surface of the condyle and those portions of the glenoid fossa and articular eminence, against which the condyle, together with the interposed meniscus, normally rests and moves, are all normally covered with cartilage arranged in layers. Fig. 1. The innermost, or that layer of cartilage immediately adjacent to the subchondral bone, is hyaline which shows varying degrees of calcification, while the outermost zone is fibrocartilage, consisting of many fibers running parallel to the surface. This cartilaginous arrangement is consistent with all weight and stress-bearing joints, making them capable of withstanding considerable compression. Resilience is one of the prime characteristics of articular cartilege. It is very elastic to frequent intermittent pressure, while continuous compression tends to slow the rate of recovery to its original shape. The mechanism of this elastic action apparently consists of squeezing out and reabsorbing fluid. This action is important in the nutrition of the cartilage because of its avascularity. The articular cartilage is one of the most primitive tissues of the body and lacks both vascular and nervous tissue and possesses a very low metabolic rate, in consequence of which it has but limited reparative ability. The respiration of cartilage is almost nil. The metabolic rate is only one-tenth that of connective tissue,

apparently because of the great preponderance of matrix over cells.

No nerve fibers have been demonstrated in either the subchondral bone or articular cartilage, but there is an abundance of sensory and vasomotor nerves in the synovial membrane, capsule and periarticular tissues, which apparently are the receptors for pain. These same mixed nerves innervate the adjacent muscle and other soft tissue of the area. This close nervous connection between the joint structures and the muscles controlling the motion of joints was worked out as long ago as 1863 by Hilton and known as "Hilton's Law." It postulated that the same nerve trunks that supply the joint also supply the muscles moving the joint, and, in instances, the skin area over the insertion of these muscles. Joint pain results from any agent irritating these free nerve endings, whether from inflammation and increased pressure in the tissue or from trauma.

As far as the temporomandibular joint is concerned, most cases of arthritic involvement fall into one of four major groups:

(1) Traumatic, or those resulting from acute direct trauma to the joint.

(2) Infectious arthritis—the frankly infectious cases caused by specific organisms.

(3) Rheumatoid arthritis, which is of unproved etiology, but probably due to collagen degeneration.

(4) Degenerative joint disease, or those cases of degenerative changes incidental to age and other factors.

It is these various types of arthritis acting, singly or in combination, that accounts for the great majority of internal derangements of the temporomandibular joints.

### *Traumatic Arthritis*

Only acute direct trauma is included under this designation. Table 1. In the past, chronic joint disease produced by minor, repeated trauma, and continued over a long period of time, has been included in this term. However, since the end result in the latter may differ in no way from degenerative joint disease, practice excludes such cases and labels them simply as degenerative joint disease secondary to trauma. Acute traumatic affections of the temporomandibular joint may result from strains, severe blows or falls, fractures into the joints, dislocations, and hemarthrosis; in fact, any severe trauma to the region. The effect of trauma upon articular structures depends on the type, severity and duration of the injury, as well as the reaction of the individual. The primary pathologic reaction is a synovitis, but this is often complicated by hemorrhagic effusion into the joint space which is especially true when the capsule is torn. When articular structures other than the synovial membrane are damaged, changes similar to degenerative joint disease result almost invariably.

Traumatic joint disease is usually monarticular. The symptoms, usually acute with sudden onset, include pain, particularly on motion, and localized tenderness. Objectively there is localized tenderness, swelling, limited motion, frequently ecchymosis and muscle spasm. Without synovial effusion, hemorrhage, or both,

into the joint space the area will appear negative to roentgen examination, but, with effusion or hemorrhage into the joint area, it will appear cloudy and indistinct.

TABLE 1

*TRAUMATIC ARTHRITIS*

| | |
|---|---|
| ETIOLOGY. | Lesions secondary to acute direct trauma. Blows, strains from mouth gags, fractures into joints, dislocations, hemarthrosis, etc. |
| PATHOLOGY. | Depends upon type, severity and duration of injury. Synovitis, often with hemorrhagic effusion. Capsular tears and detachment or laceration of the articular cartilage. |
| SYMPTOMS. | Usually monarticular. Pain, swelling, limited motion, muscle spasm and ecchymosis. |
| ROENTGEN FINDINGS. | Negative (early) unless cloudy and indistinct from effusion of synovia and blood. |
| TREATMENT. | Rest. Cold compresses over area followed by heat. Aspiration of fluid. |
| PROGNOSIS. | Good with early and adequate treatment. |

Treatment in acute traumatic disease of the temporomandibular joint consists of rest, cold applications in the early stages, followed by heat in the later stages and the administration of analgesics for the control of pain. Jaw action should be limited, resorting to intermaxillary immobilization if necessary, but passive action should be permitted as the pain and soreness subside. The prognosis is good as the disease is reversible with proper and adequate treatment. A specific history of trauma, sudden onset of symptoms and its monarticular nature serve to establish the diagnosis and differentiate it from other joint derangements.

### *Infectious Arthritis*

This form of arthritis has reference to the invasion of the articular structures by specific organisms. Table 2. It may be caused by several mechanisms:

(1) Direct wounds of the joints;

(2) Blood borne injection;

(3) Direct extension of infection from neighboring areas.

The metastatic form of infectious arthritis is the most frequent and may follow septicemia, pneumonia, gonorrhea, meningitis and influenza, to name a few. The temporomandibular joint is especially exposed to infectious processes by direct extension from the mandible, maxilla, mastoid and the facial cervical area. The

pathologic changes vary in intensity with the type of organisms and the resistance of the individual. Early symptoms include an acute onset of fever, chills, sweats, leucocytosis, pain, muscular spasm and joint tenderness. The onset of this disease is usually acute, but may be insidious, frequently polyarticular and migratory at first, finally settling in one or more joints. The cardinal signs of inflammation are usually present and pain is often severe. The synovial fluid may become purulent and, if the disease is not arrested, it causes destruction of the articular structures and crippling. It is one of the most frequent causes of ankylosis of the temporomandibular joints.

TABLE 2

*INFECTIOUS ARTHRITIS*

| | |
|---|---|
| ETIOLOGY. | Joint invasions by specific organism either by direct introduction, extension from neighboring areas or by metastasis. Incidence markedly reduced by antibiotics. |
| PATHOLOGY. | Varies from mild synovitis to extensive joint destruction dependent upon the type of organism and efficancy of treatment. |
| SYMPTOMS. | History of primary infection. Generalized symptoms of acute infection, limitation of motion with pain and swelling of joint. |
| ROENTGEN FINDINGS. | Synovitis followed by destruction of bone and cartilage. Hypertrophic changes. |
| TREATMENT. | Antibiotics. Selection determined by gram strain and culture. |
| PROGNOSIS. | Good. Ankylosis a terminal possibility. |

The existence of specific infectious arthritis is usually suggested by the history. Bacteriologic tests should be resorted to whenever possible, and demonstration of the organisms in aspirated joint fluids allows for a proven diagnosis. The incidence of this type of arthritis has decreased greatly since the advent of chemotherapy and the antibiotics. Treatment is directed both symptomatically and locally. The affected joint should be at absolute rest, as motion is extremely painful. Passive motion should be allowed as soon as painless movement is possible. Analgesics for the relief of pain are used as indicated. The invading organism should be identified at the earliest possible moment and the most effective antibiotics determined by gram stain and culture. Treatment early in the disease is of paramount importance to minimize destruction of joint tissues.

### *Rheumatoid Arthritis*

The cause of this type of arthritis is not fully understood. Table 3. It is a disease of temperate climates, with low incidence in the tropics. It has a definite familial

tendency. Females are more commonly affected than males in a ratio of 3:1. Eighty percent of the cases occur between the ages of 20 and 50, with the peak at 35 to 40. Investigation shows that in the period preceding the onset of symptoms, emotional shocks are common and severe depression is frequently present. Pregnancy causes an amelioration of symptoms in a significantly high proportion. Recent work indicates that adrenal cortical function in rheumatoid arthritis is normal. Incidentally, these hormones have been instrumental in dealing the final blow to the theory of an infectious etiology of this disease. Evidence is accumulating that the initial lesion of rheumatoid arthritis involves, primarily, the interfibrillar substance of the connective tissue.

Rheumatoid arthritis produces widespread pathologic lesions. It is a generalized disease in which the joints are the main scene of destruction but structural changes are found in many additional tissues and organs. Symptoms referable to the skeletal system are muscle stiffness, joint stiffness, aching, pain and swelling. With

TABLE 3

*RHEUMATOID ARTHRITIS*

| | |
|---|---|
| ETIOLOGY. | Unknown. Initial lesion suspected as being located in the interfibrillar substance of the connective tissue. |
| PATHOLOGY. | Destruction of joints. Lesions of muscles, tendons, nerves and visceral organs. |
| SYMPTOMS. | Many prodromes with a variety of clinical forms. Fatigue, emotional upsets, fever, joint and muscle pains. Disturbed function and structure of organs. |
| ROENTGEN FINDINGS. | Fusiform swellings. Skeletal atrophy. Destruction of articular structures and deformity. |
| TREATMENT. | Systemic with supportive therapy to joints and body. Physiotherapy, psychotherapy, X-ray, Gold salts, ACTH, cortisone and hydrocortisone. |
| PROGNOSIS. | Guarded. Ankylosis a complication. |

progression there is generalized decalcification and destruction of the joint structures that may lead to ankylosis. The temporomandibular joint may become involved but it is rare that it is the first joint to become involved and it is usually associated with generalized rheumatoid arthritis. The management of rheumatoid arthritis is an involved problem, varying with the conditions that are met in each particular patient. Measures of proven value are rest, physical therapy, psychotherapy, orthopedic care, chrysotherapy, Roentgen therapy, ACTH, cortisone, and hydrocortisone. Being a generalized disease, treatment must be diverse with regard to both the body and mind. Exacerbations and remissions are the rule and they may be mild or severe. The prognosis, at least to the present time, must be guarded. In 5 to 10 percent of cases the disease continues or progresses despite all treatment.

### *Degenerative Joint Disease*

This is a process associated with the physiologic aging of the tissues of joints, aggravated by strain, oft-repeated microtraumata and deformities, the end result depending on a summation of these factors. Table 4. It occurs chiefly in older individuals and, for the most part, is unaccompanied by systematic manifestations. It is characterized, pathologically, by destruction and hypertrophy of the joint structures, and, clinically, by pain made worse by use and relieved by rest. Joint aging makes its appearance in the second decade of life and is universally present after the age of forty but only about 5 percent of the people are clinical victims of the disease. Other theories have been advanced as etiological factors in this disease since similar changes are seen in joints where functional stress is not present. Undoubtedly, some of these theories are partial factors in the explanation of certain individual cases. Symptoms begin usually in the fourth and fifth decades. If they begin early in life excessive stress and strain on the joint usually are the forerunners.

TABLE 4

*DEGENERATIVE JOINT DISEASE*

| | |
|---|---|
| ETIOLOGY. | Physiological aging, microtraumata and possibly unknown constitutional factors. Malocclusion and abnormal occlusal habits are major factors. |
| PATHOLOGY. | Softening and destruction of articular cartilage with degenerative changes of disc. Proliferation, spurs, lipping, osteophytes and deformity of the condylar head. Hypertrophy of synovial villa. |
| SYMPTOMS. | Local. Insidious onset. Pain, limitation of motion and stiffness. No systemic manifestations. |
| ROENTGEN FINDINGS. | Narrowing of joint space. Sharpening of articular margins. Deformity. |
| TREATMENT. | REST. HEAT. Correction of abnormal functional mechanics. Analgesics. Hydrocortisone. |
| PROGNOSIS. | Good. Ankylosis not a complication. |

The movements, positions and functional stress of the temporomandibular joints are dependent on muscular function and the interocclusal relationship of the upper and lower teeth. Deviations from the normal in occlusal dental relations and in muscular physiology exert abnormal stress and strain on the joint structures. This chronic irritation, combined with the normal aging of joint tissues having a low metabolic rate and poor reparative ability, is the cause for the majority of temporomandibular joint disturbances and, accordingly, will be dealt with in more detail.

*Etiology*

The temporomandibular joint is only a part of the masticating mechanism. The teeth, muscles of mastication, ligaments and other less important structures combine to form this complex system. The joints and musculature are the unadaptable parts of the mechanism. The form of the joint is established early in life and the only changes thereafter will be degenerative. Muscles do not shorten or lengthen except as a result of disease or by surgery. The teeth and their supporting structures then are the changing and adaptable parts of the mechanism and it is to them that attention must be focused in establishing a probable cause of the disease.

Functional analysis of malocclusion is an involved study. It includes evaluation of the path of closure of the mandible from the rest position to the occlusal position, occlusal relations in lateral and protrusive movements, the amount of free way space or interocclusal clearance and abnormal functional habits. In the movement of mandibular closure premature contacts of certain teeth may occur, from which point the mandible will be directed from its normal to an abnormal path of closure, ending in a position of displacement. This is evidenced by a horizontal glide of the mandible from the point of initial contact to the occlusal position. Bilateral and protrusive movements of the mandible, with the teeth in occlusion, will point out cuspal interference. Periodontal changes should be observed in this connection as a tooth or teeth in traumatic occlusion invariably incriminate themselves both clinically and roentgengraphically. Incisal and occlusal attrition, as well as loss of teeth, and other factors that contribute to overclosure with the development of increased free way space permitting of abnormal joint excursion, should be evaluated. Excessively deep vertical overlaps may be caused by overeruption of the anterior teeth, others to the undereruption of the posterior teeth, or a combination of both. Interference in the performance of the masticatory stroke with all types of malocclusion directs strains of varying degrees on articular structures of the temporomandibular joints.

The nervous habit of grinding the teeth maintains severe compression on joint structures for long periods of time. The teeth suffer severe wear of their incisal and occusal surfaces as the result of this habitual grinding, thus causing greater free way space, permitting additional strain on the joint structures. When these stresses extend beyond the limits of tolerance degenerative changes occur.

*Pathogenesis*

The initial demonstrable lesion is a softening, roughening and the formation of horizontal cracks in the superficial layer of the articular cartilage and the formation of fissures which fill with blood and undermine the cartilage to separate it from the subchondral bone. Fig. 2. The early degenerative changes are most noticeable at sites subjected to the greatest pressure and movement. As the disease progresses, the articular cartilage becomes structureless and the subchondral bone surface becomes irregular and broken. Fig. 3. Ultimately, portions of the cartilage may be worn away completely, exposing the underlying bone. Such exposed bone becomes dense, polished and eburnated. Marginal proliferations,

referred to as spurs, lipping, osteophytes and exostoses, and deformities of the condylar head eventually occur. These changes exert a damaging influence on the articular disc and they undergo the same degenerative changes as the articular cartilage. Cracks, fissures and calcification are seen and areas of necrosis develop over exostoses. Fig. 4. The extent of degeneration varies greatly among individuals, with some showing more degenerative articular changes at 35 than others do at 60. Systemic factors may be more contributory than is generally recognized.

*Symptoms*

The symptoms of degenerative joint disease are local in character. The onset is insidious. The cardinal symptom is pain, which occurs on use and is relieved by rest. Stiffness and limitation of motion after prolonged periods of rest are common complaints. Such stiffness is of relatively short duration in contrast to the joint stiffness of rheumatoid arthritis. There are no specific laboratory abnormalities. Sedimentation rate, blood count, urinalysis and blood chemistry are normal. Lowered basal metabolic rates are found no oftener in this group than in other individuals of corresponding age. These cases are particularly responsive to slight trauma that would not produce symptoms in the normal joint. A diagnosis of traumatic arthritis in such cases is in error since the degenerative changes were present before the injury initiated the symptoms.

*Roentgenographic Findings*

One must be extremely careful in evaluating roentgenograms of the temporomandibular joints in degenerative joint disease. In some cases the roentgenographic appearance is normal, while in others many gradations of abnormality may be noted. In the early stages of the disease structural changes are limited to the cartilage which cannot be visualized roentgenographically. The most commonly observed change noted in roentgenograms is that of joint narrowing owing to degeneration and disappearance of articular cartilage. Following this, small osteophytes appear which originate from the edges of the articular surface and are evident on the roentgenograms as irregular projections extending outward from the joint margins. Fig. 5. Inasmuch as the detail of all roentgenograms of this joint is somewhat obscured by superimposition of the bones of the skull, the abundance and extent of these outgrowths are seldom fully appreciated in the roentgen study.

*Treatment*

From a pathologic standpoint this disease is chronic and cannot be cured. Since worn or damaged cartilage regenerates poorly, at best, and since osteophytes cannot be resorbed, such changes, once established, are irreversible and permanent. Nevertheless, much can be done to relieve symptoms and to prevent, or at least retard, progression of the pathologic conditions.

One of the most important points in treatment is to reassure the patient that the disease is annoying but not serious; that it is not a crippling arthritis. No obvious deformity results from degenerative joint disease as far as the temporomandibular joints are concerned.

Fig. 1—Normal articular cartilage of mandibular condyle separated by joint space from disc. X 96.

A: Innermost layer of hyaline cartilage containing large basophilic cartilage cells.

B: Second or middle layer containing fewer cartilage cells housed in lacunae.

C: Third or outermost zone, which is wider and composed of fibrocartilage, consisting of many fibers running parallel to the surface.

D: Normal articular disc composed of collagenous fibrous tissue and containing few scattered chondroid cells.

Bauer, W. H.: Am. J. Path. 17:129, 1940

Fig. 2—Early degenerative joint disease.

A: Horizontal crack filled with blood and fragments of calcified cartilage.

B: Bone marrow space is opened and its contents transformed in a fibrous tissue.

C: Fibrocartilage showing cracks and fissures and a slightly uneven surface.

Bauer, W. H.: Am. J. Path. 17:129, 1940

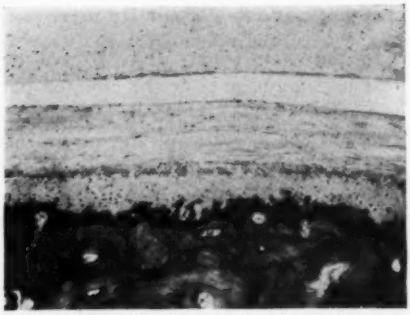

Fig. 3—Advancing degenerative joint disease.

A: Articular cartilage becoming structureless. Subchondral bone surface becoming irregular.

Bauer, W. H.: Am. J. Path. 17:129, 1940

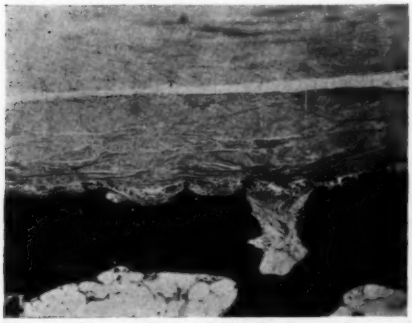

Fig. 4—Dystrophic calcification, cracks and fissures of articular disc in advanced degenerative changes of the temporomandibular joint.

Bauer, W. H.: Am. J. Path. 17:129, 1940

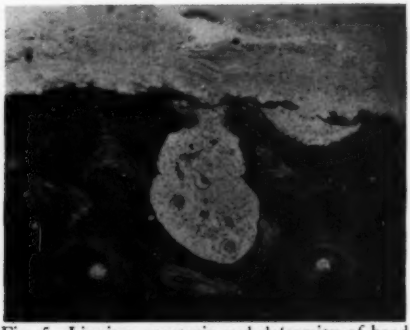

Fig. 5—Lipping, exostosis and deformity of head of mandibular condyle in advanced degenerative joint disease.

Bauer, W. H.: Am. J. Path. 17:129, 1940

The treatment of each case must be individualized. A joint with degenerative changes cannot endure much stress or strain; therefore, rest is of primary importance in the treatment of the disease. Complete rest by immobilizing the mandible is unnecessary and seldom of therapeutic value, but if the joint is forced to perform normal function, degeneration continues and may be accelerated. The patient should be allowed to use the jaw passively and to tolerance, but not to the extent to cause pain. Many patients are obsessed with the idea that their joints will become stiff unless they exercise them continuously; this is not true and the patient should be reassured in this respect.

All factors which tend to cause abnormal or excessive stress or strain on the joints should be corrected. Premature contact of certain teeth within the usual inter-occlusal clearance that directs the mandible from the normal to an abnormal path of closure must be corrected by selective grinding or extraction of teeth. Excessive free way space is treated by dental restorations and splints. The nervous type of individual should be questioned regarding bruxism or other nervous habits affecting normal mandibular uses.

In the early stages of treatment most patients benefit symptomatically from heat and massage over the affected part. Heat may be applied in any form, but best results are obtained by diathermy supplemented by other methods at home. This has the effect of producing an increased blood supply to the part. Massage can be helpful as a follow-up to heat treatments, but it should be avoided, or at least used very gently, if the area is tender. Irritating the tender tissues covering osteophytes may increase the sensitiveness of the area.

Analgesic drugs are helpful in controlling pain but should be used cautiously. Undue damage may be caused in a diseased joint by masking pain. Sodium salicylate and compounds of this drug with Phenacetin are less expensive, safer and frequently more efficacious than most other drugs of this type. Narcotics are seldom, if ever, required to relieve the pain of degenerative joint disease.

Roentgen therapy is of questionable value. If tried, 100 to 150 r, two or three times weekly for a total of three to twelve treatments is recommended. The effect of such therapy probably is analgesic only, without much, if any, effect on the disease itself.

For the past four years hydrocortisone acetate (hydrocortone) has been used for intraarticular injection in a large number of patients with different types of rheumatic disease. Remarkable benefit was noted in a large majority of patients with longer relief in joints with degenerative changes than in those with rheumatoid arthritis. In addition to giving almost immediate relief from pain and discomfort in the joint, it allows for early correction of occlusal disharmonies and other abnormalities of the masticatory mechanism. The effects of this hormone must be considered suppressive and not curative. It should never be used in cases of infectious arthritis.

Hydrocortisone belongs to the series of hormonal substances obtained from extractions of the adrenal cortex. It is a white, crystalline powder that is only slightly soluble in water.

The mode of action of hydrocortisone acetate in suppressing inflammation is not known but the speed of response would appear to place it at the connective tissue level. The result of the administration is suppression and not elimination of the pathologic state. Other steroid compounds do not have the same anti-inflammatory effect for local injection as does hydrocortisone.

Significant improvement is usually achieved within twenty-four hours after the intraarticular injection. Tenderness decreases and range of motion increases markedly. The injection can be performed as an office procedure as no elaborate preparation is necessary. Sterile precautions should be observed. The drug should be placed within the joint cavity for maximum effect but accidental injection in the peri-articular tissues is not harmful but negates its effectiveness. The use of a local anesthetic in connection with the injection is recommended.

Injection of irritating substance into the joint to produce adhesions has been reported by some therapists as giving good results. This mode of therapy seems illogical in degenerative joint disease as it produces an inflammatory reaction in tissues with which it comes in contact, in consequence of which additional injury is introduced to an early damaged joint. It is of interest to know that injection of irritating substances for the purpose of fixing recurrent, dislocating semilunar cartilages of the knee was once used but is now discarded as being ineffective and not entirely harmless.

*Surgery*

Where corrective and supportive treatment has failed to control the pain to the extent that the patient can live with the affection, surgical intervention is indicated. The purpose of arthrotomy (the surgical opening of a joint) is to remove the diseased or damaged tissues that are giving rise to pain or interfering with the normal mechanics of the joint. Since the meniscus undergoes the same degenerative changes as does the articular cartilage, menisectomy is carried out as the first part of the joint surgery. With the meniscus completely and cleanly removed the articular structures can be examined for loose bodies, osteophytes or hyperplastic synovial folds which, in turn, are removed. Postoperative joint function is begun as soon as consistent with wound healing.

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# THE MEANING OF PSYCHOSOMATIC MEDICINE

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When words are used with a multitude of meanings there arises the danger that they will have no meaning at all. Such is the problem with the term "Psychosomatic Medicine," and the purpose of this presentation is a brief discussion of the present meaning of the term in mid-twentieth century medicine.

As to the word itself, it seems to have been used in the German psychiatric literature of the early nineteenth century, although not in the sense that we use it today. Heimroth employed it in a paper which set out to prove that all mental illness originated from sin. There also was a "somatogenic" school in nineteenth century Germany which diligently observed certain physiological minutia of pulse, respirations, etc., in relation to emotional disturbance, but failed to relate them to an interdependence of mind and body. Even well before 1800 there was pertinent thought on the importance of an individual's thinking, feeling, and doing in relation to his disease. John Hunter made mention of "passions" as factors in the descriptions he wrote of his own anginal symptoms, and Sydenham made an observation that about two-thirds of the diseases of his day were fevers and of the remaining third he felt that probably half were what he called "hysteric passion." Even though at the beginning of the nineteenth century the organization and use of medical knowledge were significantly conditioned by concepts of mind-body relationship, the actual expression of such concepts were handicapped by the fact that while the medicine of the body had appreciably liberated itself from theological and moral restrictions in its freedom of investigation and speculation, the medicine of the mind was still largely bound to religion and dogma.[1]

Psychosomatic medicine in the modern sense is firmly bound up with and dependent on dynamic psychiatry, and this did not truly emerge and clear itself of some of the restrictions mentioned above until the twentieth century. A phrase that is used in the understanding of comparative anatomy and embryology is that ontogeny recapitulates phylogeny. This is an erudite way of expressing the idea that an organism in its growth passes through the evolutionary states that have brought it to its present development. One might consider that medicine has progressed (phylogenetically, if you will) through certain great phases and that the medical education (or ontogeny) of the individual medical student tends to recapitulate it.

The first great "phase" of modern medicine was that of anatomy. In the fifteenth and sixteenth century, Vesalius and Paré, utilizing observation and experience, contributed understanding of the body's structure. In the next great "phase" which spans and follows the life of William Harvey, the functions of the body were first understood, and physiology became a part of medical understanding. Following the invention of the microscope by Leeuwenhoek, attention was focused on pathology and bacteriology, and all emphasis was on finding an etiology for

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disease. The late nineteenth century ushered in the concept of bed-side teaching and the conviction that the physician could only learn of the patient from talking with him and examining him. With the discovery of Salvarsan early in the twentieth century, the great age of chemotherapy began—an age we are still in, to some extent. Also has come intensive technological advance with sensitive equipment for registering objectively more and more of the body's functions in health and disease. So far, the "phases" of medicine as outlined above fairly closely paralleled the standard instruction given to the medical student. He begins with anatomy, learning the body's structure. He then progresses to physiology and understanding of its function. Next is pathology and a study of the morbid changes produced in structure and function by disease. And after his pre-clinical period he is instructed in the art of history taking and the practice of physical examination and is heavily saturated with the details of technological advance. This phylogenetic progress of medicine has led to greater general knowledge, but has also meant greater specialization and a narrower scope of inquiry for the individual doctor.

There is cause to speculate that later day historians may find our present moment in medicine the beginning of another great turning point with two major new emphases. One is on rehabilitation and a better understanding of the aging process. The other is on a better understanding of emotional function in relation to disability. It is this latter emphasis, which seems to this writer to constitute the true meaning of psychosomatic medicine, which could be defined as an approach to medicine characterized by the conviction that what goes on in the mind and in the emotions influences the way in which the body functions and may cause or influence the course of physical disease.

Three major factors seem to contribute to this new emphasis and they may be stated as follows:

1. New understanding of unconscious motivation is derived from the systematic psychoanalytic study of some thousands of patients over 50 years. This has given meaning to a host of previously unrelated and inexplicable clinical phenomena. (This orientation for an understanding of dynamic unconscious processes is the chief distinction between the "intuitive" family doctor and the modern psychosomaticist.)

2. Scientific studies of animals and man have demonstrated that profound disturbance in body functions may result from emotional stress. These studies would range from the observations of Beaumont on Alexis St. Martin to the comparable observations of Wolf and Wolff on Tom—the modern day Alexis.

3. Experiences of World War II brought thousands of physicians into contact with the way in which severe disturbances of both social behavior and bodily function developed as a result of emotional stress.

The "war neuroses" of World War II were no different from the "shell shock" of World War I, but there was a different impact on medical thought. With orientation to the psychosomatic point of view, such patients provided dramatic validation of the power of unconscious motivation and often new understanding

of previous clinical experience. The disappearance of symptoms under appropriate psychotherapy, and, even more important, the new insight into the interrelation of mind and body gained by many physicians in military service made it clear that the psychosomatic approach provides an invaluable tool for the practice of medicine in all its branches. Many such physicians sought additional training in psychiatry and psychosomatic medicine after discharge.[2]

Today, in marked contrast to twenty-five years ago, most doctors realize that a large fraction of all who seek medical advice suffer from emotional stress and conflict which play an important role in precipitating, intensifying, or prolonging their symptoms and disability, and which cannot be ignored in diagnosis or prognosis and plan for treatment.

Today it is well known to informed lay public as well as to physicians that emotional factors—either wholly unrecognized by the patient or, at best, poorly understood—are operative not only in "nervous breakdown," where no disease can be detected, but also in many well established disease processes, such as peptic ulcer, asthama, arthritis, hypertension, glaucoma, diabetes, menstrual disorders, impotence, and even infertility, epilepsy, thyroid disease, colitis, obesity and many skin disorders.

The discovery and management of the emotional factors pertinent to an illness require considerable special training for the physician. In psychosomatic medicine the point of departure is the emotional life of the patient and especially some understanding of the unconscious processes which are inevitably a part of all personality function. One needs a knowledge of normal personality development as a frame of reference and with it an idea of what constitutes appropriate responses to various problems and stresses.

Also, one needs an attitude of sympathy and permissiveness. This may have to be cultivated tangentially to conventional medical education where the combination of knowledge of basic science plus good history taking ability and technique of physical examination presumably will guarantee a diagnosis. Modern medical education seems to be taking these emphases into consideration in curriculum planning. More and more of the students' time is being allotted to systematic study of the concepts mentioned above.

*SUMMARY:*

The concept that man's state of mind and state of physical well being are intimately related is not new.

Systematic investigation in animals and man plus a greater understanding of unconscious processes have put this concept more on a scientific and less on an intuitive basis.

As the present teaching of medicine parallels the way that medical knowledge has developed, increased interest and emphasis may herald a new phase in medicine.

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# SUCTION TUBE FOR FISTULAE

## ESPECIALLY FOR ESOPHAGEAL DUODENAL AND PANCREATIC USE

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External fistulae of the gastric intestinal tract, despite great advance in surgical technic, still occur with sufficient frequency to command attention. The management of fistulae of the stomach, biliary tract, jejunum, ileum and colon has been fairly well standardized and the response to definitive surgical procedures is usually excellent. Modern aggressive surgical attack upon the oesophagus, duodenum and pancreas has brought with it an increasing number of postoperative fistulae of these organs. Here the problem is vastly different than with fistulae of the remainder of the gastrointestinal tract. In the first place definitive surgical intervention usually is impossible and for this reason reliance has to be placed on conservative measures of treatment. Furthermore, the problems created by the loss of fluids and electrolytes and the inability to ingest sufficient nourishment to maintain the patient in nutritional balance often have proved to be insurmountable. Indeed, not too much time has elapsed since the patient with a duodenal fistula rarely recovered and the patient with an oesophageal fistula practically always succumbed.

In our experience the present day high incidence of recovery from these distressing postoperative complications has been due largely to the maintenance of nutrition made possible by the administration of whole food through a polythene tube passed well beyond the fistulous opening.

The local fistulous tract itself also requires treatment and it is with this phase of management that this presentation deals. During the past six years we have used an especially devised inexpensive suction tube which has helped greatly in caring for these patients.

The suction tip introduced into the fistulous tract must be in contact with the site of origin of the fistula and should be soft and non irritating. Glass, hard rubber, or metal tubes are not practical because of the possibility of pressure necrosis and because they may cause discomfort if the patient changes position. Suction at the origin of the fistula is most desirable because in this way the fluid, which often is irritating, is removed promptly, thereby enabling the fistulous tract to fill in with granulation tissue. The tubes are prepared in the following manner.

In a twenty-inch length of plastic intravenous tubing two small openings are cut at 2 cm. intervals about ten inches from the end of the tube. These openings extend through one half of the circumference of the tube. The tube is then folded on itself so that the two small openings are in apposition. The limbs of the tube are united proximal to the opening with a single fine silk suture. (Fig. 1.) Suction is applied to one free end of the tube and air is allowed to pass in through the other. In certain instances owing to mucus, necrotic material, feces, etc., the secretion

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Fig. 1—Holes are cut about 2 cm. apart on the same side of plastic i.v. tubing. The tube is then folded on itself bringing the holes in apposition with each other. The tube is kept in this position by sewing with fine silk. Various sizes of tubes may be used as desired.

Fig. 2—For routine use and especially in cases where the fluid is thick a third tube is added. The additional tube may be used for a slow drip of fluid if desired.

becomes too thick to be aspirated. In these cases a second single tube with an opening about 1 cm. from the distal end is sutured to the previously described tubes so that all the openings are in close proximity. (Fig. 2.) This in effect provides three tubes, one for suction, one for air and one for a slow drip of saline or water to dilute the secretion sufficiently to allow it to be aspirated.

After preparation in the foregoing manner these tubes are gently inserted into the fistulous tract and the suction tip is placed near the origin of the fistula. The proximal ends are then taped to the skin. Care must be taken to be certain that the tube for the inflow of air is kept open. As these tubes are pliable and transparent they can be easily removed for cleansing by the injection of warm water or by means of a fine probe or stylet. This is more frequently necessary where some necrotic tissue is present. All of the material removed is measured and recorded and when indicated is strained and returned to the gastrointestinal tract through small polythene jejunal tubes. These suction tubes have been used in the treatment of fistulae of the oral cavity, oesophagus, stomach, duodenum and jejunum, biliary tract, pancreas and colon with excellent results. Collection of the fistula secretions in this manner gives the fistulous tract a better chance to heal, the skin is kept dry and the patient's nursing problems are simplified. The fluid and electrolyte balance is more easily maintained since all fluids may be returned to the gastrointestinal tract when indicated. The tubes are soft, readily available on every hospital floor and quite easily constructed.

# PUNCH PRESS INJURIES TO THE HAND

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The hand and the punch press are natural antagonists. Encounter between these two have only one constant feature. The punch press always wins.

These machines are very carefully designed and built to incorporate safety features. It is difficult to understand why the workmen constantly risk their livelihood by misusing these devices. However, in an individual area such as this, the punch press injury is a very common occurrence. The injuries range from finger tip amputations through all types of injuries to forearm amputations.

This discussion is limited to the so-called massive hand injury. Massive hand injury can be defined as that injury including more than the fingers and in which there is loss of some portion or damage to all the major components of the hand, i.e., skin, tendons, muscles, bones and nerves.

When a patient with this type of injury is seen, the problem is not what has been lost, but what remains that can be made useful. It is important to realize that parts still viable are not necessarily parts that can be made useful. Fingers, for example, that are crushed and battered, may survive. They may survive, however, only to become a rigid prong interfering with the use of the hand. Thus, they are an impediment and should be removed at the time of original injury.

The difference between a part that is merely viable and a part that can be made useful is indefinable. It can be judged only by the experience of the operator and careful consideration of the occupation of the injured man.

The following cases will illustrate the problems encountered and the compromise with nature that has been accomplished.

The first case is that of a machine operator who reached into a running machine to dislodge a malformed piece of work. Figure 1 and 2 show massive avulsion of skin of web, amputation of the terminal phalanx and one-half of the proximal phalanx of the thumb, amputation of the index and partial amputation and filleting of the middle finger. This middle finger was viable, but badly fractured with loss of its radial surface skin covering. It was therefore decided that this digit was non-salvageable.

The treatment was based on the fact that he had two good fingers and a stump of thumb, the latter having no covering. Simple skin grafting would be to no avail, as it would provide no cushion against the exposed bone. An abdominal pedicle graft was then planned and utilized. (Fig. 3.) The patient now has a soft, flexible covering with adequate subcutaneous padding for his thumb and radial side of the hand. The thumb is moveable, providing him with a very short but flexible and apposible thumb. He has returned to his usual job.

The second case is that of a man injured by a punch press. The x-rays showed

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Fig. 1

Fig. 2

Fig. 1, 2, and 3—Case 1.

Fig. 3

Fig. 4

Fig. 5

Fig. 4, 5, and 6—Case 2.

Fig. 6

Fig. 7

Fig. 8

Fig. 7, 8, 9, 10, 11, and 12—Case 3.

Fig. 9

Fig. 10

Fig. 11

Fig. 12

Fig. 7, 8, 9, 10, 11, and 12—Case 3.

Fig. 13

Fig. 14

Fig. 13 and 14—Case 4.

extensive fracture damage and amputation of his fingers. (Fig. 4.) In addition, there was avulsion of the skin of the palm, loss of tendons and small muscles of the hand. Abdominal pedicle flaps were used and after many stages, formed and thinned to produce two stubby fingers. Tendon transplants added strength to these fingers. Figures 5 and 6 show this hand with these stubby but useful digits.

Figures 7, 8 and 9 show a third injured hand with the same etiology—a punch press injury, showing massive loss of three fingers and portions of the hand itself. The x-rays show the fracture damage. The second metacarpal was shortened and the third and fourth metacarpals removed to narrow the hand. The wound was covered by an abdominal pedicle graft as in Figure 10. The final results show a functional claw-hand, Figures 11 and 12, with soft pliable skin replacing that which was lost. A tendon graft to the inner aspect of the fifth finger has added strength for opposition to the thumb.

Figure 13 shows a similar fourth case with loss of the central portion and leaving only the thumb and fifth finger. The second, third and fourth metacarpals were removed. There was adequate viable skin to affect a primary closure. Figure 14 shows a postoperative result of this hand which is a strong, grasping claw that the patient has learned to use and he has returned to his original job.

These cases show clearly the damage that can be done by these machines and illustrates dramatically that these hands cannot be repaired. Repair implies the return to the condition before injury. It is obvious that none of these hands have been brought back to anywhere near their normal condition. The result of treatment is therefore a salvage and a compromise with nature and not a repair.

# CONSERVATIVE MANAGEMENT OF DIABETIC FOOT COMPLICATIONS

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Improved management of diabetes has resulted in an increased average life span. As diabetics succeed in living with their disease for twenty or more years, an increasing number develop a severe degree of atherosclerosis. This is in part a result of survival into the age group in which atherosclerosis is present in the non-diabetic and in part due to the known tendency for longstanding diabetics to develop arteriosclerosis.

Atherosclerosis of the lower extremities has increased the incidence of diabetic foot complications including gangrene and infections. As experience accumulates in handling these complications, it is found that lesions which at first sight seem hopeless can often be treated conservatively without surgical amputation. Even before the introduction of sulfonamides and antibiotics, self-amputation of gangrenous digits could be accomplished and extensive infection of the foot could often be controlled after adequate incision and drainage. Antibiotics have removed the risk of conservative management and served to control most foot infections. We feel that most cases should be given at least a trial of conservative therapy. In a few cases, amputation will later be necessary because of intractable pain, extensive tissue destruction, or too great loss of skin. In favorable cases, at least a part of a foot will be preserved for the patient to walk on. Not only will the patient be spared the psychic trauma of losing an extremity, but the preservation of the natural foot will preclude the increased strain on the other foot, which is certain to result from ambulation with crutches or an artificial leg. It must be remembered that if the lesion is caused by arteriosclerosis, arteries in the sound leg may be involved to an equal degree and greatly increase the hazards of trifling injuries to the remaining foot. For this reason, amputation of the first foot should be avoided as long as possible in the arteriosclerotic patient. Bilaterally deficient circulation renders ambulation with an artificial limb a much more hazardous undertaking than it would be in a younger individual whose foot might be amputated for a crush injury and whose remaining foot had normal arterial circulation.

### *Prevention of Foot Complications*

Most foot lesions are preventable and have their origin in minor injuries which could have been forestalled if the patient had been aware of the hazards and had used care to avoid them.

Good diabetic control is an important preventive measure. Patients should be warned that poor control favors infection, disturbed lipoprotein metabolism,

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atherosclerosis and neuritis.

With the onset of claudication, leg cramps and cold feet, the patient should be made aware of the impaired circulation and proper foot hygiene should be re-emphasized. Patients are frequently unaware of impaired pain and temperature sensation, and are thus deprived of this important protection against various forms of trauma, heat, cold and chemical agents. Loss of pain sense may allow a noxious agent to remain in contact long enough to produce necrosis of tissue. It is for this reason that hot water bottles and self-surgery should be forbidden.

Diabetics should be told to take care to keep the skin of their feet intact. This is particularly true where the skin has become thin, inelastic and deprived of its fatty subcutaneous cushion, as a result of impaired circulation. A daily foot bath is recommended, avoiding excessive use of soap to preserve natural oil. Dryness and cracking can be avoided by oiling the skin with lanolin, baby oil containing lanolin, or petrolatum.

Patients should be warned of the dangers in the use of all forms of heat, ordinary skin antiseptics, home remedies and salves. The combined analgesic and necrosing properties of phenol render its use, even when diluted, especially hazardous and its use without medical supervision has directly resulted in loss of an extremity. Tincture of iodine is too harmful to tissues for use in the diabetic foot. Seventy percent alcohol has been satisfactory for use as an antiseptic in the home and our patients are advised to use nothing else without the permission of the doctor.

Hot water bottles, chemical heat bottles and electric pads are not to be used because of the burn hazard if pain and temperature sense is impaired.

It is our feeling that all foot care, beyond the ordinary daily hygiene, should be in the hands of a trained person. The diabetic person should not do any cutting or peeling of skin. Patients are encouraged to report at once to their doctor if anything unusual about their feet occurs. The use of adhesive tape has resulted in skin injury when the skin is thin and atrophic. The superficial layer of skin can easily be pulled away with the tape as it is removed, resulting in an ulcer. If adhesive tape is to be used, it should be applied to the outside of the dressing, or only to healthy skin.

Socks should be of ample size, both as to the length and width, to avoid binding the toes together. Socks preferably should provide a slight cushioning effect against the sole and upper of the shoe and allow air circulation.

Shoes should provide a full half inch space beyond the great toe and be wide enough to allow all toes to be moved. A last should be selected which allows the first toe to be in line with its metacarpal without pressure of the shoe corner on the great toe-nail. The lining should be smooth, free from projecting seams and hard ridges. No lining is better than a torn cloth lining. The inside of the shoe should be examined frequently with the hands to detect foreign bodies and nail points which can injure the neuritic foot repeatedly without the patient becoming aware. Soft leather has the advantage of not traumatizing the skin, but the disadvantage of exposing the toes to injury by falling objects. Hard metal toes (with ample

room) are needed where metal and heavy objects are handled. New shoes, even if they fulfill the above requirements, should never be worn more than an hour or two for the first few days. After shoes have been broken in, daily pressure on the same area can be avoided by wearing different shoes on alternate days. The patient should not walk without the protection of shoes or slippers.

The nails should be trimmed straight across, the corner thus produced may be rounded slightly, but should be outside the skin fold. If the shoes are ample in size, nothing further is needed to prevent ingrown nails. It is best that nail trimming not be done by the patient, because he must cut from an awkward position; he may cut too deep because of neuritis or impaired vision.

Corns usually result from ill-fitting shoes. They can be prevented or relieved by proper fitting shoes. They should never be trimmed by the patient or other untrained person. First, the pressure should be relieved by allowing sufficient room in the shoe. The corn may be softened by alternate soaks in water and oil applications. With the pressure relieved, the softened corn will gradually be extruded. The top of the corn may be cut off gradually, as it lifts itself out. This is best performed by a physician, nurse who understands the problem, or a co-operative chiropodist.

Callus is also a result of continuous pressure, usually to a larger area than with a corn and usually on the sole of the foot. Where callus results from disturbed foot mechanics, the orthopedist may help. Excessive pressure on the callus may be relieved by using a sponge rubber ring in the shoe (1). Calluses can be removed by relieving the heavy pressure and by advising patient to oil with lanolin, baby oil or petrolatum. After softening, the callus can gradually be pared down, if

Fig. 1—After adequate wide incision.

Fig. 2—Healed after 3½ months, showing skin graft.

necessary. The callus with its ischemic underlying tissue is a frequent site of chronic infection and deep ulcer. Healing of the ulcer cannot be accomplished until the surrounding rim of callus has been pared down by a surgeon. This operation can be facilitated by rest in bed and softening of the callus by soaking. This type of plantar ulcer is a constant invitation to organisms capable of producing cellulitis of the foot.

Another point of entrance for micro-organisms is the fissure between the toes. This may result from a fungus infection or simply from excessive moisture. Shoes which are wide enough to allow air circulation between the toes will reduce perspiration and the susceptibility to fungus infection. The skin between the toes should be thoroughly dried. Propionic-acid dusting powder ("Desenex") daily will combat moisture and act as a safe fungicide. Strong fungicides should be avoided.

*EVALUATION:*

Clinical examination alone frequently leads to an adequate evaluation of the circulation in the affected foot. On attempting to assess the prognosis of a foot complication, one must consider the patient as a whole, the degree of symptoms, the condition of the arteries generally and locally. Specific laboratory procedures may add further information. Certainly the future cannot be told by just a single cursory look at an extremity. Our plan of evaluation is given in Table I.

TABLE I. Evaluation

I. Walking capacity, degree of symptoms
II. Careful clinical examination
  A. Palpation of vessel walls and pulses
  B. Condition of skin and subcutaneous tissues
  C. Relative color, warmth
III. Results of conservative regime
IV. Other available tests
  A. Occlusion index
  B. Sympathetic blocks
  C. Arteriography

In the last analysis, the truest evaluation results from a trial of conservative therapy. We have seen many patients who had been advised to have a leg amputation respond to conservative therapy to a degree that a major amputation has been obviated.

Although atherosclerosis in diabetics is usually a diffuse process, Leriche's (1) syndrome of sclerosis of the aortic bifurcation should be considered in diabetics as well as non-diabetics when a deficient circulation occurs in the feet. Aortography has become a very useful adjunct to the evaluation of the condition of the vessels. When the sclerosis is localized, in a large artery, resection followed by a homograft offers hope of restoring circulation to the foot.

## *MANAGEMENT OF DIABETIC GANGRENE*

Gangrene which continues to progress into the foot and which produces local and systemic symptoms while under conservative supportive therapy is best

handled by surgical intervention. Conservative management should be tried in cases where the gangrene is limited to a digit or digits.

The management of diabetic gangrene is listed in the following table:

TABLE II. Management

I. Diabetic control
II. General health
III. Protection against physical and chemical injury
IV. Antibiotics
V. Vasodilators
VI. Anticoagulants
VII. Consider sympathectomy

The diabetic patient with gangrene of the foot should be put at rest. The diabetes is meticulously controlled. This means the fasting blood sugar should be between 100-150 milligrams percent; and the twenty-four hour urine should contain no glucose or less than 15-20 gms. If the patient is anemic, the condition is best corrected by transfusion. The individual who is anemic and in negative nitrogen balance resists infection poorly and heals slowly. If there is no azotemia, a good protein intake is given. Testosterone may be of value in helping put the patient in a positive nitrogen balance. If there is neuropathy associated with the gangrene, and there usually is some, parenteral vitamin B and B-12 are given.

The positioning of the patient and limb in bed is important. It is a common practice to elevate the feet, particularly if there is edema. This is probably of value in disease of the veins, but since this decreases the already short blood supply of the feet, we see that a level position is maintained with the patient comfortable. The foot is left exposed to the air and the leg is protected by a cradle which must be well padded so as to avoid further injury to the limb if the patient should happen to bump it. The foot and leg are moved by the patient frequently to prevent the development of pressure areas where the foot rests on the bed. Physio-therapy is used to keep the joints motile and aid muscle tone in the rest of the leg and body. Heat is not applied as it would increase the metabolism of the cells, thus requiring more blood which is not available.

If infection is present, antibiotics are started. By reducing the infection and swelling there may be enough improvement of the blood supply to keep the gangrenous process to a minimum. Before the day of antibiotics, the infection and swelling would cut down the blood supply and the gangrene would advance rapidly up the foot and leg.

Gangrene advances by thrombosis. When the lesion seems to be progressive and there is considerable pain, a ten to fourteen day course of heparin is started. The heparin seems to help control the pain as well as prevent advancing of the gangrene. Heparin is given intravenously 75 mg. five times per day. With this dosage there has been very little trouble with bleeding.

Engelberg and Massell (2) have used heparin in peripheral vascular disease with some benefit in walking tolerance. Waldow et al (3) have shown that heparin had a lowering effect on lipemia in normal subjects, and there was also a reduction

of the gamma globulin concentration. This suggests that a heparin-globulin reaction may be related in the clearing of lipemia.

Drug therapy is somewhat limited in cases of gangrene of the foot. There are two types of medication in use. One type tends to increase the blood flow. Among these are aminophylline, priscoline, nicotinic acid and papaverine. If some spasm is present they may be of value. The other type of drug used is one which controls pain. It is best to avoid narcotics; salicylates given at regular intervals with sedation control most of the pain. Dry gangrene is usually painless. Alcohol by mouth may be tried for its sedative and vasodilating effect.

If there is a marked degree of vaso-spasm determined by the different tests, a sympathectomy should be considered. Because the tests are not always conclusive, preliminary paravertebral block may be tried. If the foot temperature is increased, a sympathectomy is indicated. The sympathectomy, of course, will not cause a reversal of the gangrenous process, but rather keeps it from progressing and it does warm the leg and foot so that they will be more comfortable.

A case report shows the favorable outcome where amputation was previously advised: A 62-year-old female who had had diabetes for fifteen years was admitted to Henry Ford Hospital in December, 1951, for gangrene of the right foot. She gave the history of stepping on a toy block three weeks before. One week previously a larger blister appeared on the plantar surface of the foot. The blister broke and bloody pus was discharged.

The examination at this time showed an obese female with a gangrenous ulcer on the right foot. There was evidence of swelling and inflammation. The admitting temperature was 103, hemoglobin 9.7, W.B.C. 18,200 and blood sugar 154.

The patient was placed on penicillin, given blood—and a surgical debridement. On the seventh hospital day, the foot was opened widely. For thirty days saline soaks were used, alternating with streptodornase and streptokinase. After thirty days the wound had cleared and healthy granulation tissue was present. Three months after admission, the area was debrided and a dermatome skin graft was done. After three and one-half months of hospitalization, the foot had healed and the patient walked out of the hospital without crutches and with a usable foot.

## *CONSERVATIVE SURGICAL PRINCIPLES*

The decision to do a leg amputation has in the past frequently been decided upon without a real attempt to determine what can be accomplished by a lesser procedure. Certainly it is unwise to waste time and money in pursuing a conservative course in the face of insurmountable odds, for in such instances an above-knee amputation will result in rehabilitation in the shortest possible time.

Injudicious mechanical instrumentation in the local care of open foot lesions may do a great deal of harm. An active thrombosis may be precipitated in adjoining healthy tissue by attempts to separate dead tissue before the proper time. The instinctive desire to do something extensive must be curbed and only inspection and very gentle palpation with light pressure used during examinations.

The low vitality of the tissues must be always kept in mind. Tourniquets and clamps are always to be avoided and the finest concepts of plastic surgery adhered to (4).

With the conservatism permitted by the use of antibiotics, the performance of conservative foot amputations has been repopularized. Here, as in repairs after trauma, as much viable tissue as possible should be saved. As much of the metatarsals as possible should be saved, as any additional length improves the walking ability.

All pertinent phases of a patient's life and physical condition must be explored to determine the best method of treatment. The financial position of a patient and his family, his capacity for work, his ability to use a prosthesis, the degree to which he will adhere to a medical program and advice, and the condition of the opposite extremity are to be considered.

The results of a conservative attitude with the employment of all measures short of amputation have in our hands given very encouraging results in borderline cases. Antibiotics by controlling infection have removed the urgency of amputation. The principles of conservative therapy are given in Table III:

TABLE III
Conservative Surgical Principles

I. Adequate incisions for infection
II. Antibiotics
III. Trial of conservative management
IV. Debridement
V. Local amputation
VI. No touniquet or tight dressing

## *CONCLUSIONS*

1. The conservative management of diabetic foot complications gives surprisingly good results. Many patients respond to a conservative program, where major amputations were earlier considered necessary.

2. The avoidance of trauma of every type, including injudicious probing and handling, is of the greatest importance if success is to be obtained.

3. A careful program of management is essential.

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# THE INVOLVEMENT OF JOINTS OF THE EXTREMITIES FOLLOWING TRAUMA IN OTHER AREAS*

JOHN LYFORD, III, M.D.**

The involvement of joints of the extremities in relation to trauma can be divided into two sections: (1) the joints have been injured directly by the trauma, and the post-traumatic changes are, therefore, the result of that direct injury; (2) the joints have not been involved directly in the injury, and the changes in the joints are secondary to the injury elsewhere in the extremities or trunk. It is this second group in which the joint involvement is secondary to injury elsewhere in the extremities that we shall consider at this time.

The group of joint involvements secondary to injury in other parts of the extremities is impressive. It includes reflex sympathetic dystrophy, arthrofibrosis and adhesive pericapsulitis ("frozen joints"), muscle atrophy with secondary loss of motion of the joints, traumatic arthritis, limitation of motion of the joints, changes concurrent with "Volkmann's ischemia," myositis ossificans, and chondromalacia patellae.

Reflex sympathetic dystrophy, which is recognized in its most extreme form as "Sudek's atrophy," is that condition manifested by pain in the extremity, loss of motion of the joints of the extremity, atrophy and fibrosis of the muscles with atrophy of the small bones of the involved extremity, and subsequent fibrosis of the capsular structures about the joints. This is seen most frequently in the hands and feet; and may occur following even minor trauma, such as contusions, of the forearm, hand, ankle, or foot. It is a condition extremely distressing to both the patient and his doctor; and when it has reached its peak of development is often irreversible. To prevent this complication it is necessary to recognize the onset at the earliest time; and to do this one must be immediately alert to any irregularity in the symptoms of pain in the injured extremity. It is most frequent after such injuries as a "Colles's" fracture of the wrist, sprain of the anke, or fractures about the ankle joint. It manifests itself by intractable, otherwise unexplained pain in the hand or foot of the injured extremity, frequently in that type of patient who tends to be classified as a "chronic complainer," and the symptom of pain is out of proportion to that which should be expected. This pain, with swelling of the involved extremity—particularly of the fingers or toes—and some change in the temperature of the digits are early signs. Treatment is not always successful, but requires immediate and energetic use of sympathetic blocks—either cervical or lumbar for the corresponding upper or lower extremity—in association with the use of vasodilator drugs such as Priscoline, systemic Cortisone, and intensive active motion of the involved digits.

Arthofibrosis and "adhesive pericapsulitis"—commonly called "frozen joints"—are more frequent but equally distressing joint complications of contiguous

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trauma. These are seen in injuries about the cervical spine in which there is protective immobilization of the shoulder joints in relation to referred pain; in shoulder joints in which motion has been limited because of immobilization of the upper extremity by plaster casts in the treatment of fractures of the elbow, forearm, or wrist; and in knees when the lower extremity has been immobilized in treating fractures of the hip, femur, or tibia in hip spica or long leg casts. Involvement of the other joints of the extremities with this condition is less frequent. It should be prevented by as early active—and, if necessary, passive—motion of the respective joints as can be carried out without endangering the treatment of the fracture. Active abduction and circumduction exercises to the shoulder concurrent with the immobilization of the arm for the treatment of the fracture is essential; and these exercises should be continued until there is no longer any possibility of involvement of the joint. Similarly, elbow joints should be removed from immobilization as soon as treatment of the fracture permits—and this is particularly true of fractures about the wrist when short arm casts permitting active motion of the elbow should be instituted as soon as feasible. Also, finger joints should be kept actively and passively mobile throughout the period of fixation of the rest of the extremity. Following removal of the immobilizing plaster cast, intensive physical therapy to all the joints of the extremity should be carried out. In like manner, treatment of the knee to prevent stiffness of this joint from enforced immobilization should, like the shoulder, elbow or wrist, be carried out by active mobilization and physical therapy. Associated with this type of involvement of the knee joint there occurs occasionally chondromalacia patellae. This may respond to conservative measures, but if sufficiently symptomatic, may require correction by surgical procedures.

Loss of functional motion of the joints of the extremities, particularly extension of the knee, is often associated with atrophy of the musculature from disuse during immobilization of the extremity. Most frequently so involved is the quadriceps muscle mechanism in the lower extremity. This can, to a great extent, be prevented by the institution early of active quadriceps muscle strengthening exercises even while the extremity is immobilized.

In lower extremities in which a fracture of a long bone has resulted in malalignment or shortening of the bone there may result a poor weight-bearing mechanism of the adjacent joints, particularly the knee or hip joint, or the spine with a resultant traumatic arthritis developing in these joints. This is a difficult condition to prevent; but should be approached by attempting to equalize the leg lengths or to correct for the malalignment by the use of conservative measures ("lifts" to the shoe on a short leg) or the surgical correction of the deformity. In older patients with preexisting degenerative joint changes—and in individuals in any age group with preexisting rheumatoid arthritis—there may be a marked exacerbation of pain and limitation of motion from enforced immobilization of such joints associated with treatment of injuries elsewhere in the extremity. This difficult problem can be combatted only by the early, functional motion of these joints.

Limitation of motion in elbow joints resulting from mechanical malposition of

fracture fragments of the radius and ulna, with or without radio-ulnar synosteosis, preventing full pronation and supination as well as flexion and extension must be recognized at the time of initial treatment of the fracture, and prevented by correct anatomic alignment of the fracture fragments.

Involvement of elbow, wrist, and interphalangeal joints secondary to "Volkmann's ischemia" with changes in all the soft tissues about these joints is a most distressing complication of injuries of the upper extremity, and can be prevented only by recognizing the condition when it is impending and taking energetic measures to prevent progression of the condition. Such measures may require immediate surgical intervention by a skilled vascular surgeon, particularly if occlusion or rupture of the brachial artery is suspected.

Limitation of motion of the joints of the extremities, especially the elbow and occasionally the knee, by myositis ossificans traumatica developing in the soft tissues about these joints is a condition about which we know little as to the cause, and even less as to its prevention. However, this condition should not be treated by surgical measures at an early stage.

This brief outline of the involvement of joints of the extremities secondary to trauma not directly involving such joints does not cover all the possibilities; but it does indicate the frequency and scope of such complications. In the last analysis, treatment of these conditions is best carried out by bearing in mind the possibilities and preventing them whenever possible; and by recognizing them early when they do occur, and whenever possible, referring them to the specialist most experienced in these conditions in the field of orthopedic surgery and traumatology.

# NEW TECHNIQUES IN PTOSIS SURGERY

ROBERT A. SCHIMEK, M.D.*

Ptosis, or drooping of the upper lid, may be congenital or acquired. Congenital ptosis is three times more frequent.[1] The success of an operation for congenital ptosis depends on the degree of levator function and the presence of associated defects. Developmental defects frequently found with congenital ptosis are (1) paresis of the superior rectus or other extra-ocular muscles, (2) epicanthus, (3) amblyopia, (4) jaw-winking, (5) Duane's retraction syndrome, (6) blepharophimosis, and (7) mental retardation. The presence of any of these may modify the type of surgery indicated.

Acquired ptosis may be due to neurological lesions, trauma, or diseases affecting the lids. Acquired ptosis should not be operated upon until all other treatment directed to the cause has failed, and it is apparent that there will be no spontaneous improvement in the ptosis. Paralysis of the third nerve or its nucleus, or the cervical sympathetics on one side (Horner's syndrome) should usually be present at least a year before the ptosis is considered permanent enough to warrant operation. Ptosis due to cicratrix from skin defect, trachoma, or pemphigus, or from local tumors, should be treated first by resection of the scar tissue or tumor. Later a ptosis operation may be necessary. The treatment of myasthenia gravis or progressive nuclear ophthalmoplegia is usually non-surgical, since such patients are likely to develop postoperative diplopia or exposure keratitis. Other causes of acquired ptosis which usually contra-indicate surgery are: active states of palpebral edema, trachoma, and vernal catarrh; untreated syphilitic lesions; thyrotoxicosis; hysteria; intoxications from coal tar derivatives, lead poisoning, and botulism; and acute infections such as diphtheria and influenza.

The 80 odd varieties[2] of ptosis operations can be divided into four groups. They are based on the principle of (1) resection of the skin, (2) use of superior rectus, (3) use of frontalis muscle, or (4) enhanced use of the levator.

Skin excision methods may be dismissed as not being permanent or practical, except in cases of blepharochalasis. Use of the superior rectus is permissible for bilateral ptosis with normal superior rectus action. However, this type operation carries with it some danger of exposure keratitis, from interference with normal blinking and incomplete lid closure during sleep. In addition, other complications which may occur include diplopia and hypotropia, arching of the lid margin, entropion and trichiasis, and excessive postoperative scarring.

Frontalis motion can be transmitted to the tarsal border of the lid by permanent sutures, temporary sutures forming scar tracts, flaps of skin or fascia lata, and muscle strips of orbicularis or of frontalis. This type of operation gives the lid voluntary movement in the absence of levator function. The disadvantages are the peculiar facial expression which may result from use of the frontalis, the lack of

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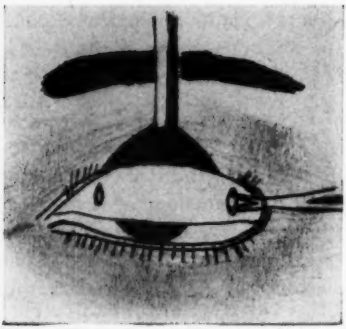

Fig. 1—The upper lid is everted with a lid holder. With sharp-pointed scissors a buttonhole is made medially and laterally through conjunctiva and levator tissue above the tarsal plate.

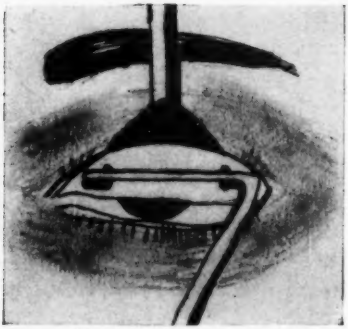

Fig. 2—One jaw of a Berke ptosis clamp is then passed into the temporal buttonhole, passed across the lid beneath conjunctiva and levator, and out through the nasal buttonhole. The jaws are clamped together.

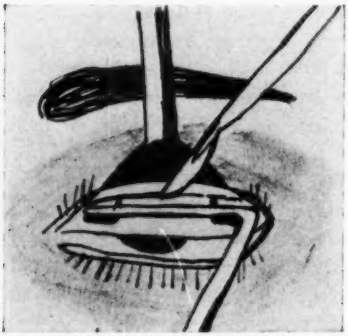

Fig. 3—An incision is made between the clamp and the border of the tarsus through conjunctiva and levator.

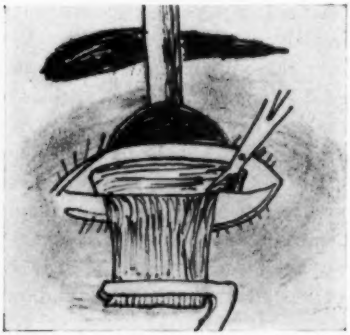

Fig. 4—By sharp and blunt dissection, the levator, Mueller's muscle, and the conjunctiva are separated as one layer from the septum orbitale, to the level of the upper fornix.

Fig. 5—A 2 or 3 mm. tarsectomy is performed.

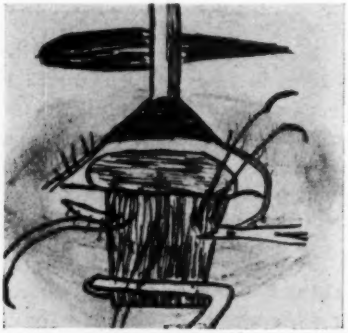

Fig. 6—3 double arm sutures are then passed through the layer of conjunctiva, Mueller's muscle and levator about 12 mm. to 15 mm. from the free border. Excision is performed 2 to 3 mm. in front of the suture line.

complete synchronization of movement between the lid and the eye when the eye is rotated vertically, and danger of exposure keratitis during the early postoperative period.

Operations which enhance levator action (as in levator resection) have the great advantage of producing normal blinking and synchronous following movements of the lid with the eye. There is a relatively small incidence of excessive post-operative scarring, lagophthalmos, deformation of the lid margins, diplopia induced by operation, or unsightly asymetrical elevation of the brow. With levator action present, levator resection gives the most ideal results. However, if there is no levator action, poor lid mobility may result.

The original Blaskovics technique of levator resection has the disadvantage of requiring tedious dissection of the conjunctiva and the levator from the other lid structures. Also the conjunctive may be buttonholed and Mueller's muscle may be accidently reflected with conjunctiva. Annoying bleeding is usually present during the dissection. A modification of the Blaskovics operation which has been used by Guyton[3] since 1946 has the advantage of quickness, simplicity, and preservation of the normal relationships of the levator, Mueller's muscle and conjunctiva to the septum orbitale. Bleeding is minimal because of the minimum dissection required. What dissection is done lies between natural tissue planes, as between levator and septum orbitale. The technique is as follows: The lid is everted. A horizontal incision is made through conjunctiva and tarsus parallel to and a few mm. from the superior tarsal border. The tarsal rim with its vessels can be clamped by a Kelly clamp or a Berke lid clamp. The rim of tarsus with the attached levator, Mueller's muscle, and conjunctiva is separated from the lid well up into the superior fornix. This tongue of levator (with its attached tarsal rim and conjunctiva) is resected and the stump is reattached to the resected tarsus.

Iliff[4] uses a procedure similar to this, grasping the levator and conjunctiva with the Berke lid clamp. This modified Blaskovics technique is described in figures 1-9.

Figure 10 is an example of a bilateral case of ptosis, which was more marked on the left side, with fair levator action on both sides. The levator on the right was resected by the original Blaskovics technique and the levator on the left was resected by the modified technique used by Guyton and Iliff. Although the same amount of levator tissue was resected in each eye, the modified technique (performed on the more ptosed left eye) resulted in a greater correction than the Blascowicz technique. Theoretically this could be expected because of maintenance of normal relations between the shortened levator muscle, Mueller's muscle, and the shortened conjunctiva with the modified technique. There has been no complication from excessive shortening of the conjunctiva in any of the cases done with the modified technique.

Many authors agree that levator function must be present preoperatively, or levator resection will give inadequate correction, or at best, correction with poor lid mobility. However, de Blaskovics[5] stated that "even when the muscle is absent—an apparent function develops even in those cases in which this muscle

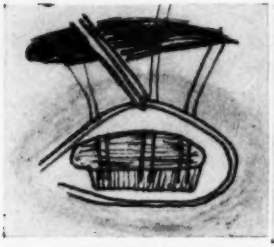

Fig. 7—The lid retractor is then removed and the upper tarsus is everted with forceps. The 3 double arm sutures are passed through the upper tarsal border and brought out about 2 mm. from the lash border.

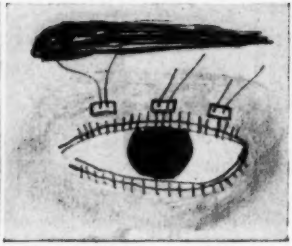

Fig. 8—These are tied over pegs or over a long thin rubber peg.

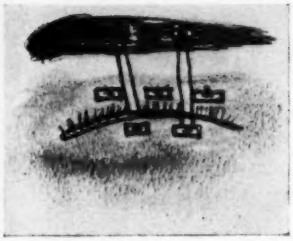

Fig. 9—Two Frost sutures are then placed from the lower lid to the brow to protect the cornea. A modified pressure dressing is applied.

Fig. 10—Preoperative appearance of bilateral ptosis, more marked left eye, with fair levator function O. U.

Fig. 11—Postoperative appearance with Blaskovics performed on the right and the modified Blaskovics technique on the more severely drooping left upper lid. The same amount of levator was resected in each eye, but the modified technique was more effective.

Fig. 12—Combined levator shortening and frontalis operation: Levator and conjunctiva are isolated on a Berke ptosis clamp exactly as in the modified Blaskovics technique. (as shown in figures 1-4) If a fairly sturdy piece of levator has been obtained, the conjunctiva should be peeled off from the distal 12 mm. of the levator tendon where it will be buried in the lid. This can easily be done with scissors by blunt dissection.

Fig. 13—The levator is split longitudinally and a double armed mattress suture passed through each end.

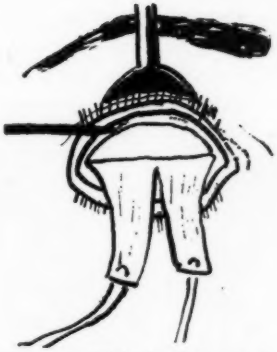

Fig. 14—With the lid everted on a retractor, small incisions are made with a knife in the tarsus just below and parallel to the superior tarsal border in the medial and lateral halves of the tarsal plate.

Fig. 15—Small horizontal incisions are made medially and laterally above the brow as in the Reese type operation. A ptosis knife is passed through each brow incision and down through the corresponding tarsal plate buttonhole. The sutures from each tongue of levator are passed through the hole in the blade of the ptosis knife.

Fig. 16—With the ptosis knife, each longitudinal band of levator is pulled through the buttonhole in the tarsus, up to the frontalis muscle, and out of the corresponding brow incision. The levator is sutured to the frontalis muscle through the incisions above the eye brow. These incisions are sutured. The lower lid should be pulled up to protect the cornea by Frost sutures as in Figure 9.

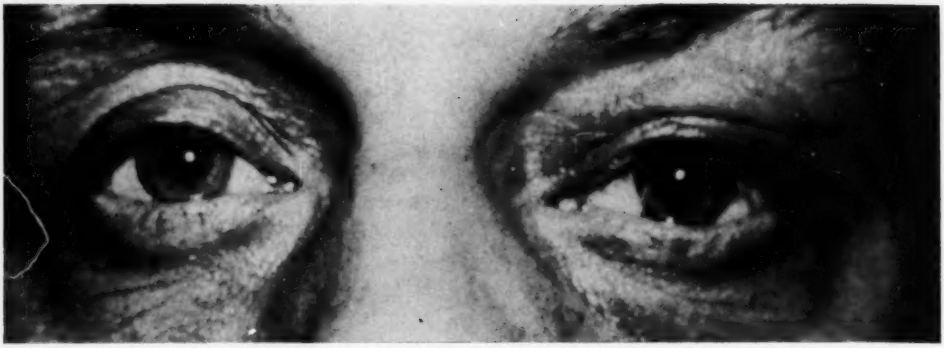

Fig. 17—Result of combined levator resection-frontalis operation, left eye. Eyes in primary position. Two weeks postoperative.

Fig. 18—Good lid movement on upward gaze with levator and frontalis action.

Fig. 19—Satisfactory lid closure.

is completely paralyzed or atrophic." Many others, such as Lindner and Jaensch, agree with this.

In cases without any levator function preoperatively, it should be advantageous to combine shortening of the levator with a frontalis type operation. One would like to enhance levator function to the maximum degree and still obtain accessory frontalis action. This accessory frontalis action can compensate for any under correction or lack of mobility of the lid that might result from doing a simple levator resection in a case with complete paralysis. Techniques combining shortening of the levator with a frontalis type operation have been described by Wilder[6] and Beard.[7]

The new operative procedure to be described herein exactly follows the modified Blaskovics technique for the stages up to the isolation and dissection of the levator muscle. (Figures 1 to 4.) Figures 12 to 16 show the subsequent stages. The levator tendon and muscle are split longitudinally into two portions. Two horizontal incisions are made through the tarsus just below its superior border. Each tarsal incision or "buttonhole" must be several millimeters from the center of the lid to avoid subsequent arching of the lid margin. Two skin incisions are made above the brow as in the Reese operation. Each tongue of levator is pulled through its tarsal buttonhole and up through the lid to the corresponding brow incision. The levator is sutured to the frontalis through the two incisions above the brow. Thus an effective sling of levator muscle and tendon is looped through the upper tarsus and is attached to the frontalis. The result gives the advantages of shortening the levator muscle and also allows use of the frontalis muscle as an auxiliary voluntary means of moving the lid.

Figures 16 to 18 show the postoperative result of a combined levator resection and utilization of frontalis type operation. It is of interest that this patient had two previous unsuccessful ptosis operations: A resection of the tarsus by the Everbusch technique had failed, and Friedenwald-Guyton suture also failed when the tantalum wire broke.

In summary, a modification of the Blaskovics procedure is recommended because of its technical simplicity, shorter operative time, and equal or slightly greater effectiveness. A new operation combining levator shortening with frontalis utilization is described for those cases of ptosis without any apparent levator function.

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# THE TOTAL AND PERMANENT DISABILITY PROBLEM

Louis J. Steiner, M.D.*

In any consideration of the problem of "total and permanent disability" there are certain fundamental principles and concepts which should attract our attention. As society has broadened its conscience and responsibility toward its components, the problem of evaluating disability has become more accentuated. The injured workman is provided medical aid under law, and insurance of various types cushions the losses due to injury and disease. Prolonged periods of disability challenge his economic, social and personal welfare. To meet this problem, before normal retirement, there have appeared in labor-industry contracts disability provisions designed to aid the individual who has made a reasonable contribution to society in terms of production and service. This type of disability compensation is also available on a personal basis through various insurance companies. In more recent years, special legislation has been passed, such as the amendments to the Social Security Act of August, 1950. The latter are Federal grant-in-aid programs.

For physicians called upon to evaluate whether or not an individual is "totally and permanently disabled" it is absolutely necessary that they understand clearly what the contracting parties had in mind, or, when the state is involved, the intent of the law and the rules for its enforcement. The physician should be fully informed since there may be benefits of considerable variation. The phraseology of the law may be confusing.

The designation "totally and permanently disabled" needs clarification, particularly if it is taken to imply that both factors must be evident with absolute certainty. Strictly interpreted, such a person would be deceased or moribund. A person who is paralyzed from the waist down may write a successful book or conduct a business by telephone. However, he will require the assistance of another person to meet many of his primary needs, and work will have to be brought to him. Ability to do occasional odd jobs can hardly be disqualifying.

The modern industrial concept behind many plans is that utter physical and mental helplessness as a result of injury or disease is not a requirement of "total and permanent disability." "Permanent" surely cannot be interpreted in its extreme sense of absolutely predictable, everlasting, unchangeable, unending disability. It is used relatively in contra-distinction to "temporary" or "transient" disability. This is apparent from the usual requirement that evidence of continuity of the disability may be required at suitable intervals. This provision recognizes the fallibility of human predictions. The problem of certainty of prediction is constant. Obviously, slight chance of improvement cannot destroy permanency, and a mere possibility of permanence cannot be enough for a finding of permanency.

Our concept, from an industrial point of view of "total and permanent dis-

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ability," is that the individual has been disabled by bodily disease or injury so as to prevent him from engaging in a regular occupation or employment for remuneration or profit; such impairment will be permanent and continuous during the remainder of his life. Many contracts deny benefits if the impairment is related to chronic alcoholism or narcotics, to injuries sustained while engaged in felonious assault or self-inflicted injuries, or to injuries suffered while in the services of the armed forces of any country. A further explanation of terms is in order.

*Impairment* means the presence of a disease process, physical or mental, or in combination, or an anatomical abnormality.

*Permanent* refers to an impairment in which the concept of "cure" cannot be applied. Certainly, any condition which medical science thinks is not likely to respond to known treatment may be considered as "permanent." Furthermore, any condition likely to remain static or to become worse unless certain therapeutic measures are carried out may be deemed to be permanent if treatment is unavailable or inadvisable.

"*Permanence*" does not rule out the possibility of vocational rehabilitation or even recovery from the impairment. Every physician knows of cases which respond favorably to treatment after an unfavorable prognosis or after the disabling condition became arrested.

Newer advances in medical treatment may change the picture of a disabling disease. However, in those cases in which corrective treatment has been attempted and has failed and the individual has submitted to further treatment with failure, there is some question whether benefits should be denied. When the individual is resistant or has lost faith because of past failures or when there is a real presence of further jeopardy, there is raised a point of reason and justice. The American philosophy is against the use of force or undue pressure. However, those providing the benefits do not intend the disabled to remain so through choice or unwillingness to submit to cure.

*Remaining capacity* for activity is important. The great majority of people have more abilities than disabilities. These are the ability or capacity to carry on usual work or gainful employment, to get to and from work under reasonable climatic conditions, to care for personal needs, and to get along with fellow workers with a minimun of tensions.

It is of great importance to evaluate the effect of the impairment on function. This is basic. An electrocardiographic diagnosis without reference to function creates hardships, anxieties, frustrations, loss of hope and the unwarranted removal of the individual from effective happy economic life.

"*Total Disability*" refers to incapacity to engage in any useful work. The time factor does not enter the definition of "totally" as it does with "permanently." A person just operated upon for a gastric ulcer is totally disabled, but medical experience knows that he may be expected back on the job in four weeks; so there is no permanence.

An individual with an amputated leg who has held a job for the past twenty-

five years certainly has a permanent impairment, but, obviously, it is not total. The latter refers to what the individual can do in the light of his impairment and capabilities.

A regular occupation means one commonly accepted as such and not one created especially for the person. Regularity or reasonable regularity of performance goes with this. Of what use to a company is a toolmaker, a regular occupation, when the toolmaker's impairment prevents him from working more than one day each week? Gainfully employed or employment for remuneration or profit means the individual can be productive for his employer and for himself, and that his job is not in the nature of a sinecure. Evidence of gradual downgrading over a period of years because of progressive disease is clear evidence of the fact that gainful employment is coming to an end.

The role of the family physician is considerable in all cases which are brought before the impartial medical examiner. With him is first recorded the sequential medical history of the case. Thus, a record is made which may shed light on the impairment. The family physician frequently has sociological data not readily available to others. His opinions are valued and respected, and he does have the responsibility of caring for the individual medically for the future. There is little doubt that the family physician is most happy when his patient is rehabilitated or returned to an effective economic pursuit. The family physician carries the responsibility, in many instances, of the family problems brought about by the change. On the other hand, the family physician is generally not experienced in the physical, mental and temperamental requirements of industrial occupations.

When the medical impairment is severe little difficulty arises, but in those cases in which the disability is borderline, and they are numerous, special experience is desirable. There, too, is some understandable yielding of the physician to the desires of the patient. Independent examiners eliminate this situation.

The opportunity for further examination of those rejected should also include the review of any additional data submitted.

The records of the plant physician and personnel are of material assistance. Time lost because of illness, nature of illness, regrading of residual abilities or capacities, reassignments may contribute much in the evaluation.

*Social factors* such as age, racial origin, language, education, training, work, marital situation, home surroundings, may have medical significance and should be considered. The significance of these factors will vary in each case.

*Reactionary factors* make disability evaluation difficult, ambiguous and variable. They involve human equations and opinions which are difficult to regulate, standardize or correlate. Here we have the loss of will to work. This may be psychological due to aging process, to frustrations, depressions, feelings of not being wanted, adverse social relations, difficult labor situations, unfavorable labor climate, etc. Often these factors are minimal, but, at times, are important determinants.

In summary, an attempt has been made to clarify the present basic concepts of the "total and permanent disability" problem as specifically applied.

# ABSTRACTS OF RECENT PUBLICATIONS OF THE PROFESSIONAL STAFF OF THE HENRY FORD HOSPITAL AND THE EDSEL B. FORD INSTITUTE FOR MEDICAL RESEARCH

*PREVENTION OF CHOLESTEROL TYPE FATTY LIVERS IN MICE BY DIETARY DIHYDROCHOLESTEROL. W. T. BEHER and W. L. ANTHONY. J. Nutrition, Vol. *52*, No. 4, April 1954.

An investigation was made of the effect of several dietary factors on total mouse liver cholesterol and beta steroids. It was found that increasing the amount of dietary cholic acid, added to a ration containing cholesterol, increased the deposition of liver cholesterol and beta steroids up to a limiting concentration. The addition of dihydrocholesterol, to diets containing cholic acid and cholesterol, significantly decreased liver cholesterol and beta steroids. If increasing amounts of cholic acid were added to a ration containing constant amounts of dihydrocholesterol and cholesterol, there were significant decreases in liver cholesterol and beta steroid concentration. Under all conditions, total liver cholesterol and beta steroid concentrations were parallel, indicating the absence of dihydrocholesterol deposition. Total beta steroids were determined by a new procedure employing the reaction of anthrone and digitonin in the presence of sulfuric acid.

*STUDIES OF ANTIMETABOLITES. I. PYRIDINE-3-SULFONIC ACID AND 3-ACETYLPYRIDINE. O. H. GAEBLER and W. T. BEHER, J. Biol. Chem. *188*, 343-49 (1951).

It is known that the toxicity of 3-acetylpyridine (3-AP) and pyridine-3-sulfonic acid is increased by nicotinic acid deficiency in dogs. The two compounds have been considered as antimetabolites of the vitamin for this reason. It was suggested that if these compounds act by blocking the synthesis of Coenzymes I and/or II or by displacing the niacinamide moiety of these substances, the urinary excretion of $N^1$ methylnicotinamide, one of the chief products of niacin metabolism, should be altered. The addition of pyridine-3-sulfonic acid to the diets of normal and niacin depleted bitches did not effect the excretion of $N^1$ methylnicotinamide or, the Coenzyme I and II content of the erythrocytes.

Feeding of 3-AP to normal bitches receiving complete or niacin deficient diets resulted in greatly increased excretion of $N^1$ methylnicotinamide. That the substance excreted in increased amount was $N^1$ methylnicotinamide was confirmed by isolation and identification.

*STUDIES OF ANTIMETABOLITES. II. 3-ACETYLPYRIDINE. W. T. BEHER, W. M. HOLLIDAY, and O. H. Gaebler. J. Biol. Chem. *198*, 573-8 (1952).

When 3-acetylpyridine (3-AP) is fed to dogs, large amounts of $N^1$ methylnicotinamide (NMeN) appear in the urine. This finding might be interpreted as evidence that 3-AP, acting as an antimetabolite, displaces nicotinamide from its

nucleotides, prevents its incorporation therein, or alternatively that 3-AP is biologically oxidized to nicotinic acid and is thus, in a sense, a provitamin. These conjectures were tested by feeding 3-AP containing an excess of $C^{13}$ in the Carbonyl group. NMeN excretion increased markedly. The atom per cent excess of $C^{13}$ in the additional NMeN excreted was 59 per cent as great as in the injected 3-AP. In a comparable experiment nicotinic acid tagged with $C^{13}$ in the carboxyl was fed in amounts necessary to produce a similar increase in NMeN output. The atom per cent excess of isotope in NMeN was 46 per cent as great as in the ingested precursor. It was concluded that 3-AP is biologically oxidized to nicotinic acid to an extent sufficient to account for all of the increase in NMeN output observed after its ingestion.

*STUDIES OF ANTIMETABOLITES. III. THEM ETABOLISM OF 3-ACETYLPYRIDINE *IN VIVO*. W. T. Beher and W. L. Anthony. J. Biol. Chem. *203*, 895-8 (1953).

It has been shown that after the administration of 3-acetylpyridine (3-AP) to the dog only about 10 per cent of the administered antimetabolite can be accounted for as $N^1$ methylnicotinamide. Inasmuch as the benzene analog, methyl phenyl ketone is metabolized to methyl phenyl carbinol glucuronide and hippuric acid, and a small amount of unaltered methyl phenyl ketone excreted, one might expect by analogy to find after 3-AP administration increased urinary excretion of all the normal metabolic end products of niacin metabolism as well as excretion of methyl beta pyridyl carbinol, its glucuronide, and unaltered 3-AP. The urinary excretion of these substances was investigated after the administration of 3-AP. It was found that the administration of 3-AP to the bitch resulted in increased urinary excretion of nicotinic acid, $N^1$ methylnicotinamide, and glucuronic acid. The administration of methyl beta pyridyl carbinol resulted in an excretion pattern similar to that observed in the case of 3-AP metabolism. No urinary methyl-beta-pyridylcarbinol or 3-AP could be detected after the administration of these substances. Between 20 and 30 per cent of injected 3-AP and methyl-beta-pyridyl-carbinol could be accounted for by increased urinary excretion of $N^1$ methylnicotinamide and nicotinic acid.

*STUDIES OF ANTIMETABOLITES. IV. THE METABOLISM OF 3-ACETYLPYRIDINE *IN VITRO*. W. T. Beher, S. P. Marfey, W. L. Anthony, and O. H. Gaebler. J. Biol. Chem. *205*, 521-5 (1953).

To determine the site of metabolism of 3-acetylpyridine (3-AP) both *in vivo* and *in vitro* studies were necessary. It was demonstrated that the metabolism of 3-AP by the adult bitch is not altered by feeding sulfathalidine. Canine blood cells metabolized 3-AP, while blood plasma was inactive. Canine liver and kidney slices rapidly metabolized 3-AP, while muscle slices were inactive. The metabolism of 3-AP by canine liver and kidney slices was inhibited by $2 \times 10^{-3}$ M iodoacetic acid. $2 \times 10^{-3}$ M cyanide did not inhibit the metabolism of this substance in liver but was effective in kidney. Homogenates of liver, kidney, and muscle did not metabolize 3-AP. These findings permit the suggestion that oxidation of 3-AP to nicotinic acid may involve systems requiring Coenzyme I, and that this accounts for the increased toxicity of 3-AP during niacin deficiency.

PRIMARY AMYLOIDOSIS IN THE LOWER RESPIRATORY TRACT. DONALD S. BOLSTAD. Ann. Otol., Rhin., & Laryng. 63:200, 1954.

Eighteen previously reported cases of amyloid deposition in the lower respiratory tract have been reviewed. Ten of these cases were proved only at autopsy and the eleventh and twelfth only after pneumonectomy. In none of these cases was there evidence of amyloid deposition elsewhere in the body and they all must be assumed to be instances of primary amyloid deposition. A nineteenth case is added to this category and its progress recorded.

ACTH AND CORTISONE IN SURGERY. BROCK E. BRUSH. Recorder of the Columbia Medical Society, March 1954.

When a new medicinal or hormonal agent is introduced it usually goes through phases of evaluation. ACTH and cortisone, like antibiotics, should never be used as a substitute for well proven conservative principles of surgery. These hormones should never be administered in the presence of an undiagnosed abdominal condition. The greatest application of ACTH and cortisone is in the crisis of acquired hypersplenism and thyrotoxicosis. Acute non-suppurative thyroditis, anogenital pruritis and orthopedic conditions are well established indications for their use. ACTH and cortisone are being evaluated for many surgical diseases and a clearer picture of their indications and contraindications will be possible with further experience.

INDICATIONS FOR, AND RESULTS OF GALLBLADDER SURGERY IN PATIENTS OVER 65 YEARS OF AGE. BROCK E. BRUSH and FRANK ZELLER, Jr. J. Am. Geriatrics Soc. 1:863, 1953.

Many patients with so-called "silent" gall stones have been advised by the medical profession that there is no need for surgical treatment and that they will probably not have trouble in the future; "just follow a diet and be careful." We have reviewed 100 consecutive operations on the biliary tract in patients over the age of 65, in an attempt to evaluate the various procedures which are available and to determine what may be expected from surgical treatment in this age group. Elective gallbladder surgery is preferable to awaiting the inevitable complications arising from untreated cholelithiasis. The increase in morbidity and mortality caused by the complications of gallstones is such that all physicians should urge cholecystectomy at an early and opportune time, before a serious situation develops. Cholecystostomy is the procedure of choice in the acutely ill and poor-risk patient with gallbladder disease. It should be used when it is necessary to operate on a patient with acute cholecystitis in whom complete and satisfactory visualization of the entire biliary tree has been rendered impossible by the inflammatory process.

TOXICITY OF ISONICOTINIC ACID HYDRAZIDES IN PULMONARY TUBERCULOSIS; TOXICITY OF ISONIAZID AND IPRONIAZID USED ALONE AND IN COMBINATION WITH STREPTOMYCIN OR P-AMINO-

SALICYLIC ACID. E. O. Coates, Jr., G. L. Brickman, and G. M. Meade. Arch. Int. Med. 93:541, 1954.

Information available at the present time concerning the toxicity of isoniazid (isonicotinic acid hydrazide) has shown it to be a relatively safe agent for the treatment of pulmonary tuberculosis. Side-effects encountered among 42 patients receiving iproniazid and 35 patients given isoniazid show the former drug to be clearly the more toxic. Combinations of iproniazid with streptomycin or with p-aminosalicylic acid (PAS) appear more toxic than iproniazid used alone. Termination or interruption of therapy, or reduction in dosage because of toxicity, was necessary for 45% of those receiving iproniazid and only 9% of those taking isoniazid. While the majority of reactions to these compounds, particularly isoniazid, are minor, potentially serious effects include (a) mental changes, (b) peripheral neuropathy, (c) severe muscular spasms, and (d) hemoptysis. Attention is directed to one case of encephalitis and one sudden death, possibly related to administration of iproniazid. "Withdrawal" symptoms have been observed in 64% of 14 patients whose iproniazid therapy was abruptly terminated; these were not relieved by substitution of isoniazid. Isoniazid has proved to be a safe drug for therapeutic use. Iproniazid is considered too toxic for general use.

*SUCCESSFUL TRANSPLANTATION OF AN APPARENTLY BENIGH NEOPLASM. Robert H. Foulkes. Science 119:3082, 1954.

A spontaneous mammary pericanalicular fibroadenoma developed in a female albino rat of the Sprague-Dawley strain. Grossly the tumor was a hard, white, homogenous, lobulated, encapsulated, palpable mass. Six months after complete removal, no recurrence was indicated. Some investigators consider benign tumors incapable of further growth by transplantation. Small pieces of the present tumor, 2 ml thick by 3-4 ml in diameter, were implanted subcutaneously along the milk line of albino female rats. Of the transplants, 4 of 4 in the first, 5 of 6 in the second, 6 of 8 in the third, and 3 of 4 in the fourth generation continued to grow into large tumors. Increase in the size of transplant was not evident for 4-7 wk after transplantation. If no increase was evident after 8 wk, no growth occurred later. Histology of the tumors remained the same in each generation.

*CONJUGATION OF BENZOIC AND PHENYLACETIC ACIDS DURING NITROGEN STORAGE INDUCED WITH GROWTH HORMONE OR TESTOSTERONE PROPIONATE. O. H. Gaebler, W. T. Beher, and E. M. Crigger. Am. J. Physiol. *172*, 152-6 (1953).

In female dogs, the relative amounts of benzoate metabolized by the two routes involving peptide linkage with glycine and ester linkage with glucuronic acid were not altered by growth hormone or testosterone propionate, and the latter hormone also failed to influence the course of metabolism of phenylacetate in this respect. These hormones were investigated because they produce nitrogen storage in which formation of peptide linkages is involved.

PREPARATION AND STERILIZATION OF BLOOD PLASMA. F. W. HARTMAN, G. LOGRIPPO and A. R. KELLY. Am. J. Clin. Path. 24:339, 1954.

Chemical inactivation of viruses has been demonstrated in blood plasma without material changes in the plasma proteins. The most effective virucides have been found to belong to the following classes of compounds: (1) mustards, (2) imines, (3) conjugated unsaturated ketones, (4) alkyl oxides, (5) acetophenones, (6) alkyl sulfates and (7) lactones. Certain of these compounds, when added to plasma, are rapidly converted by hydrolysis to relatively nontoxic end-products. To date, the screening of approximately 400 chemicals for antiviral action in plasma has yielded only 7 that seem acceptable. Of these, beta-propiolactone shows the most promise from the standpoint of virucidal activity, biochemic properties and toxicity.

THROMBOCYTOPENIA AND ABNORMAL BLEEDING IN MULTIPLE MYELOMA. THOMAS N. JAMES, RAYMOND W. MONTO, and JOHN W. REBUCK. Ann. Int. Med. 39:1281, 1953.

During the course of multiple myeloma most patients exhibit a tendency to bleed. One would expect a megakaryocytopenia in a disease characterized by anemia of the myelophthisic variety. A study was undertaken to review the occurrence of thrombocytopenia in a series of 58 histologically proved cases of multiple myeloma. Megakaryocytopenia is consistently present in the marrow of patients with multiple myeloma. Thrombocytopenia occurs in the majority of patients with multiple myeloma and is a major cause of abnormal bleeding in myeloma patients.

TONSILLECTOMY AND ADENOIDECTOMY; A RE-EVALUATION OF RESULTS. JOSEPH A. JOHNSTON and THOMAS W. WATKINS. J. Pediat. 44: 127, 1954.

During the past thirty years there has been an increasing conservatism in arriving at the decision to remove tonsils and adenoids. While this has been accentuated by the advent of chemotherapy, the trend was evident even before that. In our own clinic, whose composition would reflect that of an average office practice in the middle economic stratum, the ratio of tonsillectomies to outpatients fell from 1:20 in 1936 to 1:50 in 1952. Results were appraised as to the effect on frequency of upper respiratory infection, abdominal pain, bronchitis, sinus infection, asthma, appetite and nutrition, and recurrent vomiting. From a follow-up study of 598 cases, and from a review of several conditions thought to be influenced by tonsillectomy and adenoidectomy, it is concluded that the procedure remains a highly beneficial one in the properly selected case.

METASTATIC TUMORS OF THE BRAIN AND THEIR RELATION TO PRIMARY AND SECONDARY PULMONARY CANCER. EDWIN M. KNIGHTS, JR. Cancer 7:259, 1954.

The importance of careful study of the lungs in patients having symptoms of cerebral neoplasm has long been recognized. The lungs are often involved by

metastasis from tumors in other organs, and primary pulmonary tumors are frequently the site of origin for cerebral metastases. A review of more than sixty-five hundred autopsies performed at this hospital, plus a study of surgical specimens, reveals that the brain was secondarily involved by malignant tumors of various types in ninety-four cases, either by metastasis or extension. Fifty-three per cent of pulmonary cancers in which the necropsy included study of the brain were found to have cerebral metastases. There is increasing evidence that a solitary cerebral metastasis, once considered a contraindication to a definitive surgical attack upon the primary site of growth, may occasionally be removed successfully along with the primary, with resultant prolongation of the life of the patient.

*REMOTE VALVE FOR DISPENSING COLLOIDAL$^{198}$ AU SOLUTIONS. L. E. Preuss. J. Scient. Instruments 31:29, 1954.

Calibration and dispensation of colloidal$^{198}$ Au solutions are frequently carried out by remote pipetting and by pressure transfer in plastic tubing through which its progress may be observed. Such a method is described.

INFECTIOUS MONONUCLEOSIS AND GUILLAIN-BARRE SYNDROME; REPORT OF 3 CASES. M. Raftery, E. E. Schumacher, Jr., G. O. Grain, and E. L. Quinn. Arch. Int. Med. 93:246, 1954.

The occurrence of nervous system involvement as a complication of infectious mononucleosis has been recognized with increasing frequency in recent years. Despite its low incidence, nervous system involvement ranks high among the causes of death in infectious mononucleosis. The occurrence of nervous system involvement as a serious complication in a small percentage of cases of infectious mononucleosis is stressed. Three such cases are reported, in which the neurologic manifestations took the form of an acute polyradiculoneuritis. Sheep hemagglutinins were demonstrated in the cerebrospinal fluid in one case. The heterophile agglutination test in the diagnosis of infectious mononucleosis is discussed, and some difficulties are mentioned. The nature of the Guillain-Barre syndrome is discussed briefly.

THE INTERNATIONAL, LANDSTEINER OR ABO BLOOD GROUPS. J. W. Rebuck. Am. J. Clin. Path. 24:266, 1954.

Suggested procedures for direct grouping of the ABO system of human blood groups are presented. Test-tube, slide-well and flat-slide methods comprise the direct grouping procedures. Confirmatory tests for checking the direct methods are also presented in detail. The latter include: reverse blood grouping; repetition of direct procedures with serums from additional sources; utilization of anti-AB serum; and subgrouping of A and BA. Several tables depicting the interrelationships of the various ABO systems of antigens and antibodies are presented. A discussion has been given of the possible effects on the methodology of grouping the ABO system of (1) use of immune grouping serums; (2) transposition of procedures utilized in the detection of unrelated blood groups and (3) recent in-

vestigations into the basic nature of the ABO antigen-antibody system.

CYTOLOGY OF THE TUBERCULIN REACTION IN HUMAN SKIN WINDOWS. JOHN W. REBUCK and J. LEWIS YATES. Tr. Nat. Tuberc. Ass., 1953.

An abstract from the Am. Rev. Tuberc. 69:216, 1954 embodying substantially this same material was published in the Henry Ford Hospital Medical Bulletin 2:39, 1954.

FUNCTIONAL AND HISTOLOGICAL FINDINGS AFTER OBLITERATION OF THE PERIOTIC DUCT AND ENDOLYMPHATIC SAC IN SOUND CONDITIONED CATS. HAROLD F. SCHUKNECHT and ROBERT S. KIMURA. Laryngoscope 63:1170, 1953.

This experiment was designed to study the effect on hearing and on inner ear morphology, of obliterating the periotic duct and endolymphatic sac. Operations to obliterate these structures were performed on a series of seven sound conditioned cats. Destruction of the endolymphatic sac failed to create cochlear dysfunction or histological changes in the membranous labyrinth. Surgical blockage of the periotic duct was associated with a moderate rise in Reissner's membrane in two ears. At the present time we do not believe that the evidence from our work and that of Uyamo prove that blocking the periotic duct causes dilation of the endolymphatic system. Tests of cochlear function will be done by the method of recording evoked strychnine spikes from the auditory cortex in response to the onset of pure tones.

RESECTION OF AORTIC BIFURCATION AND REPLACEMENT WITH HOMOLOGOUS GRAFT FOR ANEURYSM. D. EMERICK SZILAGYI and JOHN A. HEMMER. J.A.M.A. 154:751, 1954.

Treatment by resection of the aortic bifurcation and grafting is a very recent addition to the methods of management of abdominal aortic aneurysms. A report is given of a case of atherosclerotic abdominal aortic aneurysm that was treated with resection of the aortic bifurcation and replacement by homologous graft. During a period of observation of eight months, the result of the operation remained excellent.

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